FEEDING BEHAVIOUR IN TERM AND PRETERM INFANTS

by

Cathy Craig

Doctor of Philosophy

1997
DECLARATION

I hereby declare that this thesis was composed by myself and that the work within is my own.
ACKNOWLEDGEMENTS

The research in this thesis was funded by a Medical Research Council studentship.

Firstly I would like to express my sincerest thanks to Dr. John Amoore of the Medical Physics Department in the Royal Infirmary, Edinburgh. Without his kindness and expert advice I would never have been able to collect the data that I have presented in this thesis. In connection with the mechanical side of things I would also like to thank Jimmy Duncan for his help and support.

Prof. Dave Lee must also be sincerely thanked for the guidance and thought provoking discussions he abundantly provided over this period of study. Thanks to Dr. Ian Laing for his clinical expertise and to Madeleine Coleman for her advice and calming words in those moments of despair. Yvonne Freer must likewise be acknowledged for her invaluable assistance with subject recruitment and her discerning advice on the clinical aspects of data collection.

Lastly, but by no means leastly, I would like to thank Adrien for his encouragement and support throughout the last three years.
# Table of Contents

**Acknowledgements** .................................................................................. iii  
**Table of Contents** ......................................................................................... iv  
**Abstract** ........................................................................................................ vi  

## Chapter 1. Infant Feeding

1.1 OVERVIEW .............................................................................................. 1  
1.2 CO-ORDINATION OF FEEDING ............................................................... 4  
1.3 FEEDING PROBLEMS ............................................................................. 6  
1.4 PRETERM INFANTS ................................................................................ 7  
1.5 NEUROLOGICAL ABNORMALITIES IN PRETERMS ............................... 9  
1.6 ENVIRONMENTAL INFLUENCES .............................................................. 11  
1.7 RESPIRATORY FUNCTION ..................................................................... 12  
1.8 PHYSIOLOGY OF RESPIRATION .............................................................. 14  
1.9 REGULATION OF BREATHING ................................................................. 15  
1.10 PRETERM RESPIRATORY PROBLEMS .................................................. 17  
1.11 FEEDING PROBLEMS WITH BPD INFANTS ......................................... 18

## Chapter 2. Movement Control

2.1 CO-ORDINATION ....................................................................................... 21  
2.2 MOTOR CONTROL ................................................................................... 22  
2.3 TAU THEORY .......................................................................................... 23  
2.4 TAU-COUPLING ...................................................................................... 25  
2.5 TAU-GUIDES .......................................................................................... 25  
2.6 OVERVIEW OF THESIS ......................................................................... 28

## Chapter 3. Method

3.1 BACKGROUND .......................................................................................... 30  
3.2 SUBJECTS ............................................................................................... 32  
3.3 DETAILS OF FEEDING EQUIPMENT ....................................................... 33  
3.4 PROCEDURE ............................................................................................ 39
CHAPTER 4. SUCKING CONTROL IN TERM INFANTS

4.1 SUCKING PRESSURES .................................................. 42
4.2 CONTROL OF SUCKING ................................................... 44
4.3 CALCULATING AN INTRINSIC TAU-GUIDE ....................... 46
4.4 TERM RESULTS .......................................................... 48
4.5 DISCUSSION .............................................................. 52

CHAPTER 5. SUCKING CONTROL IN PRETERM INFANTS

5.1 PRETERM FEEDING ....................................................... 54
5.2 PRETERM RESULTS ....................................................... 56
5.3 SUMMARY OF Z-SCORES .............................................. 61
5.4 MOTOR DEVELOPMENT ASSESSMENT .............................. 63
5.5 CONCLUSIONS ........................................................... 66

CHAPTER 6. BREATHING AND SUCKING RATES

6.1 INTRODUCTION ........................................................... 68
6.2 CALCULATION OF MEASUREMENTS .............................. 70
6.3 BREATHING REGULARITY .............................................. 73
6.4 RATE OF BREATHING ................................................... 76
6.5 BREATHING WHEN SUCKING ....................................... 78
6.6 CONCLUSIONS - TERM INFANTS .................................. 79
6.7 PRETERM INFANTS WITH BPD ..................................... 80
6.8 PRETERM BREATHING REGULARITY .............................. 81
6.9 PRETERM BREATHING RATES ....................................... 86
6.10 PRETERM BREATHING WHEN SUCKING ......................... 88
6.11 CONCLUSIONS - INFANTS WITH BPD ......................... 91
6.12 SUCKING RATES ....................................................... 92
6.13 CONCLUSIONS - SUCKING RATES ............................... 94

CHAPTER 7. SUMMARY AND DISCUSSION

7.1 SUCKING CONTROL ....................................................... 96
7.2 INTERNAL TAU-PACEMAKER ...................................... 96
7.3 PRETERM SUCKING DEFICITS ..................................... 98
7.4 BREATHING REGULATION .......................................... 99
7.5 CONCLUSIONS .......................................................... 100

REFERENCES ................................................................... 101

APPENDICES .................................................................. 111
Abstract
Recent advances in technology have resulted in more preterm infants being kept alive. These increases in survival rates have, however, been met with a parallel increase in morbidity rates. My thesis has been concerned with monitoring the feeding behaviour of preterm infants born at a low gestation and birthweight, who are at risk of developing neurological problems. These infants often have difficulty co-ordinating the different mechanisms of feeding, namely sucking, swallowing and breathing.

Particular attention was paid to sucking, viewing it as a precocious motor skill. By using a new dimension of the tau theory of motor control, namely the intrinsic tau-pacemaker model, normal sucking control was established by examining the intra oral sucking pressures of twelve term newborns. The results supported a strong coupling between the tau of the pressure generated inside the mouth and an intrinsic tau-pacemaker. Six preterm infants born at less than 30 weeks gestational age and classified as neurologically at risk, were also tested from when they started bottle feeding, and for a period of four weeks thereafter. Their sucking pressures were analysed in the same way, and were individually compared to the standard set by the newborn terms. Large deviations from term norms were hypothesised to be indicative of neurological abnormalities. Irregularities in sucking control were found, but as expected, the extent of the variation differed between infants. Follow up movement assessments, when four of the infants were greater than six months corrected age, were performed by a physiotherapist. The
physiotherapist's assessment of motor development at this stage appeared to reflect the findings obtained for the infants' sucking control.

Breathing measurements were also recorded, and modulations in the pattern during feeding were noted. Again a newborn term pattern was established, and preterm infants were compared. As before, all the preterm infants tested had differing degrees of respiratory difficulty. Breathing problems were evident from significantly lower levels of oxygen concentration in the blood, and a significant difference in the timing and depth of the breaths during the sucking and pause periods of feeding.

In conclusion an analysis of feeding behaviour appears to present a wealth of information about the neurological and physiological development of very preterm infants.
1.1 OVERVIEW

Feeding is one of the most precocious skills evident in the newborn. A few hours after birth, infants are able to co-ordinate successfully the three major components required for feeding, namely sucking, swallowing and breathing. It is vital that neonates can perform such a skill, so that adequate nutrients are ingested for normal growth and development. An inability to feed effectively would lead to failure to thrive, and could have serious consequences for the newborn.

Careful integration and control of a complex array of muscle groups via the central nervous system is critical for the successful co-ordination of the suck, swallow and breathe mechanisms (Coulter and Danner 1987). The function and mechanics involved with each of the individual feeding components are outlined below.

Sucking

Sucking is a sophisticated motor skill that is responsible for the instigation and control of the flow of milk into the mouth. As foetal sucking has been observed in utero as early as the 15th week of gestation (Ianniruberto and Tejani 1981), it is hardly surprising that newborns are so highly adept at sucking successfully post partum. Several cineradiographic studies in the 1950s were able to capture on film the
actual mechanics of the tongue and jaw movements when infants were bottle feeding (Ardran, Kemp et al. 1958a). Needless to say the dangers of irradiation have prevented further use of these techniques, with real time ultrasonography (Smith, Erenburg et al. 1985) acting as a less informative but safer substitute. In spite of this, however, there is some common agreement about the processes that actually take place when an infant is bottle feeding.

When the feeding nipple is placed in the infant's mouth, the tongue surrounds the lateral sides and the bottom of the nipple and the labial and facial muscles contract to form an airtight seal. The normal cross section of the artificial nipple is circular, but once it is pressed against the hard palate and the tongue in the mouth, it forms an ellipse. The jaw then lowers and the tip of the tongue moves forward to the front of the mouth. The central part of the tongue caves in to form a hollow, creating a larger area inside the oral cavity and allowing freer movement of the tongue along to the base of the nipple (see Fig. 1.1A).

Figure 1.1
An illustration of the different processes taking place during sucking in a newborn infant.

A. Increasing Suction

B. Decreasing Suction

jaw lowered  tongue forward & down

jaw raised  tongue back & up
This enlargement of the cavity causes the intraoral suction to increase, thus aiding the flow of milk from the feeding vessel into the mouth (Bu'Lock, Woolridge et al. 1990). Once the desired peak suction pressure has been attained, the tongue starts to flatten, the jaw starts to rise and the mid frontal part of the tongue lifts to form a bell shape. This bell shape moves along the nipple in a peristaltic wave to express remaining remnants of milk from the nipple, and to propel the expressed milk to the back of the mouth where it will thereafter be swallowed (see Fig. 1.1 B). During this time the intraoral suction pressure becomes less negative. Precise synchronisation between the jaw, tongue and various other oromotor muscles is therefore essential for effective sucking to take place (Eishima 1991).

Swallowing

A swallow conveys the milk from the mouth to the stomach, but blocks off entry to the trachea and the lungs at the same time. It requires very precise integration of three different components. The first is the oral phase which is voluntarily controlled by the infant, and occurs when the milk has been propelled to the back of the mouth and has accumulated in the valleculae. The infant begins by moving the pharynx backwards and the larynx forwards, to defend its airway, so the milk can safely pass by. This process is quickly followed by the second stage where the bolus of liquid moves involuntarily through the pharynx to the oesophagus, by a peristaltic wave. This peristaltic wave continues to move the bolus along the oesophagus to the stomach, in the third and final stage of the swallow (Bosma 1994). Several studies have used stethoscopes and microphones to record the changes in the sound of the bolus of liquid as it moves along through the various stages. This technique, known as cervical
auscultation, has been very useful in trying to identify problems that may occur in the swallowing process (Vice, Bamford et al. 1995).

Breathing
During the sucking and swallowing phases it is essential that the infant maintains an adequate level of ventilation to keep sufficient amounts of oxygen in circulation. Breathing during feeding is made more difficult, as it is continually interrupted with swallows. The infant has to be more active in the control of the breathing, so it can intersperse swallows with breaths while continuing to suck. (A more comprehensive review of the control of breathing can be found later in this chapter).

1.2 CO-ORDINATION OF FEEDING
It can therefore be seen that in order to feed successfully, it is necessary to control the timing of all the different feeding mechanisms. Problems have, however, arisen when trying to quantify what efficient feeding co-ordination actually means. Some studies have documented that perfect feeding co-ordination is a 1:1:1 relationship between sucking, swallowing and breathing (Halverson 1944; Bu'Lock, Woolridge et al. 1990). This is perhaps too simplistic an explanation, as some of the factors that influence sucking, and as a result swallowing and breathing, have been overlooked. For instance, when the size of the holes in the feeding nipple were changed, it was observed that the infant altered its sucking pattern to compensate for these changes in milk flow (Mathew 1991). The infant would, therefore, wait until there was a suitably sized bolus of milk at the back of the mouth, before interrupting the breathing pattern with a swallow. This might therefore result in a 3:1 ratio for the sucking and swallowing, but would still be an efficient means of feeding, with
breathing only being interrupted for a swallow when necessary. The infant can therefore employ these various ventilatory sparing strategies to maximise oxygen input during feeding (Al-Sayed, Schrank et al. 1994). Frequency and continuity of sucking has also been found to vary if the sucrose content of a solution is changed. An increased concentration of sucrose solution resulted in infants sucking much faster (Crook and Lipsitt 1976), thereby altering the ratio of swallows and breaths.

Co-ordination between the different feeding components may also change over a given feeding episode. Mathew, Clark et al. (1985) have described two different sucking stages that occur during a feeding session. At the start of a bottle feed the infant engages in a continuous sucking phase that lasts between 30 seconds and several minutes. Following this initial period, feeding becomes more intermittent, with the infant pausing for several seconds after a short burst of sucking. Although the frequencies of the sucks in both periods were found to be similar, the pressures generated within the mouth varied significantly. These different pressures result in changing volumes of milk in the mouth, which in turn alters the swallowing frequency. Possible explanations for these different feeding stages have included the idea that the infant is receiving sensory feedback information from the satiety centres, or that the infant is reventilating following the previous period of frequent swallowing that was necessary during the continuous sucking phase. An alternative theory proposed by Kaye (1977), suggests that the pauses in the intermittent phase of feeding have evolved to allow for interaction between the mother and the infant.
It can therefore be seen from the above literature that different factors will affect sucking, and as a consequence, swallowing and breathing. This implies that the infant is capable of adapting the timing of the different feeding components to meet changing environmental and physiological needs. It does not appear that measuring of ratios will give an indication of how well an infant is co-ordinating the various systems.

1.3 FEEDING PROBLEMS

Problems do however arise when an infant cannot synchronise the timing of the various feeding components or when the mechanics of some or all of these parts break down. These problems can be physically or neurologically based. For instance, an inability to form an airtight seal around the teat, as is the case for infants with cleft palate, results in an inability to feed (Mathew 1991). Likewise damage to the motor cranial nerve nuclei or the upper motor neurons can lead to global incoordination of the feeding system (McBride and Danner 1987). Various neurological conditions are characterised by dysfunctional feeding ability. A condition known as pseudobulbar palsy for example, results in a hyperactive gag reflex and excessive tongue thrusting, which makes sucking and swallowing extremely difficult. This condition in infancy may lead to spasticity and choreoathetosis of the limbs in early childhood, a condition commonly classified as cerebral palsy. These children continue to experience problems co-ordinating their oromotor facial muscles and instigating swallows. The characteristic excessive drooling, that was once thought to be the result of hypersalivation, is also a manifestation of poor oral co-ordination. Connected to the above factors, speech production can often prove to be a problem for children with
cerebral palsy, which again can be attributed to a lack of oral motor functioning (Lespargot, Langevin et al. 1993).

A congenital disorder known as oral-buccal apraxia also seriously disrupts the organisation of the movement of the oral buccal musculature. Again there are serious feeding problems at birth including inability to control lip and tongue movements. When older, the children find it difficult to articulate speech and tend to drool excessively. (McBride and Danner 1987).

An inability to co-ordinate the oral motor muscles required to successfully control sucking and swallowing may therefore be indicative of existing neurological problems. A closer examination of feeding ability in newborn infants may give clinicians more of an insight into the infant's neurological integrity. A particular population of newborns more at risk of neurological problems are infants of extremely low birthweight and/or gestational age. These infants also experience more respiratory problems as a consequence of their preterm birth. This again will have implications for their ability to feed successfully. The next section will therefore outline the problems associated with premature birth and examine the implications for the development of the preterm infant.

1.4 PRETERM INFANTS

Causes of Preterm Labour
The search for the cause of preterm labour is still presenting a major challenge to obstetricians and gynaecologists. However, recently a wealth of evidence has been pointing towards the role of silent
intrauterine infections in the spontaneous delivery of some preterm infants of less than 30 weeks gestational age (Andrews, Goldenberg et al. 1995). Winkler and Rath (1996) have suggested that the presence of an intrauterine infection disrupts the fine balance of cytokines that exists in utero, which causes the premature production of prostaglandins, precursors to parturition. It is therefore possible that the cytokine network has a very sensitive regulatory mechanism, whereby environmental influences, such as an infection, and other intrauterine influences, such as excessive extension through multiple births, may lead to a disruption in this fine balance and cause a natural termination of the pregnancy.

Similarly, Brandtnebelshultz, Saling et al. (1995) have found that the immune status of a pregnant woman may be a determining factor as to whether a pregnancy is terminated prematurely. It was discovered that pregnant women threatening premature labour had significantly lower levels of T-lymphocytes and T-helper cells than women who were having a normal pregnancy. This would suggest that this immune impairment would provide a more welcoming environment for an ascending genital tract infection, and hence put these women more at risk for going into premature labour. Another aspect of this study was the fact that 65% of the women at risk of entering premature labour, found the pregnancy very stressful, when compared to 26% of the women having a normal pregnancy. With the known connection between stress and the immune system (Rabin, Cohen et al. 1989), this might be an important factor to consider when trying to predict mothers at risk of preterm labour.
Preterm Birth

Although advances in technology over the last two decades have brought about a decline in mortality rates for preterm infants, morbidity rates have continued to rise. Surviving infants born at a low birthweight and gestational age are more at risk for long term complications associated with their preterm condition. Specific illnesses attributed to prematurity include respiratory distress syndrome (RDS), intraventricular haemorrhage (IVH) and necrotising enterocolitis (NEC). These conditions are essentially a consequence of the physical and neurological immaturity of the infant when it is born.

1.5 NEUROLOGICAL ABNORMALITIES IN PRETERMS

Brain injury in premature infants is a significant problem. Of the surviving infants born at less than 1500g, 5-15% will develop major spastic motor deficits or cerebral palsy, with a further 25-50% of infants showing milder developmental disabilities (Volpe 1994). Cerebral palsy is a generic term used to describe dysfunctional motor control. It usually results from injury to the developing brain, affecting central and/or peripheral structures. A child is classified with a particular kind of cerebral palsy according to the type of motor disturbance (McPherson, Kenny et al. 1992). One of the characteristic motor problems is that of oral motor dysfunction, hence feeding problems. Many children with cerebral palsy experience excessive drooling, due to the inability to control and initiate the oral phase of swallowing. Other associated problems include tongue thrusting, inability to form a seal with the lips and lack of control of the bite reflex (Mueller 1974).
Neurological lesions that result from periventricular leukomalacia and periventricular haemorrhagic infarction are thought to be the major cause of these spastic motor defects (Volpe 1994). Periventricular leukomalacia is essentially the death of the white matter around the periventricular region of the brain. Arterial borders and end zones are usually plentiful in this area of the cortex. However, when an infant is born at 24-28 weeks, a sufficient arterial blood supply has not yet been developed. Only a few long penetrators of the cerebral arteries exist, with very few branches, minimising the blood supply to the surrounding white matter. After 32 weeks the number of short penetrators increases dramatically, improving vascularisation to that area (Rorke 1992). The degree of ischaemia is therefore related to the extent of penetrating blood vessels, which itself is a function of gestational age. An additional factor to consider is that the cells in this area, namely the oligodendroglia, are in a process of myelination or may still be differentiating in the pre-myelination stage, and as a result are extremely vulnerable to injury (Gilles, Leviton et al. 1983). Another point to consider is the importance of a good supply of blood to the surrounding cells in the cerebral cortex. Any impairment in the autoregulation of the vascular system in the infant (due to functional immaturity), can lead to a fall in arterial pressure and cerebral blood flow. This will then deprive the cells of oxygen and nutrients and cell death will ensue (Volpe 1994). These abnormal fluctuations in cerebral blood pressure can also rupture the fragile capillaries in the brain, leading to intraventricular haemorrhage. The subsequent reabsorption of this blood can lead to the formation of cysts on the cerebral cortex, which again can have a detrimental outcome for the developing brain.
1.6 ENVIRONMENTAL INFLUENCES

When the preterm infant is born, the brain has not developed sufficiently to cope with the continuous bombardment of surrounding stimuli. It is therefore important to take into consideration certain environmental factors that could possibly influence the growth and development of very preterm infants. In the intensive care environment the infant is exposed to high degrees of surrounding noise from the various monitors of the equipment that is keeping the infant alive. Studies have shown that it is predominantly the high frequency noise of the surrounding machinery that penetrates the incubator. Lower frequency sounds of human voices are not heard by the young infant. Likewise the lights of the surrounding environment are exceptionally bright and may actually harm the developing retina and add to the problem of retinopathy of prematurity. Persistent handling of the preterm which is necessary to perform various clinical treatments, appears to be detrimental to the infant's well being: handling events are directly related to 83% of all incidents of hypoxaemia, 93% of bradycardias and 38% of apnoeas (Murdoch and Darlow 1984). Also uncomfortable procedures, such as endotracheal intubation and chest physiotherapy, result in a marked increase in cerebral blood flow and intracranial pressure, again jeopardising the infant's welfare. Lagercrantz, Nilsson et al. (1986) found that handling resulted in an increase in the production of catecholamines to levels similar to those found in stressed adults.

Persistent environmental noise also causes the infant to spend more time in REM sleep, the stage of sleep where more apnoeas are registered. Diurnal rhythms are also disrupted with continuous artificial light, although some units try and cover the cots with a sheet to try and lessen
the degree of exposure to the surrounding light, and introduce some sort of night and day patterning. The environment of the neonatal unit therefore provides unnecessary challenges for the developing preterm infants, which may complicate existing neurological problems.

1.7 RESPIRATORY FUNCTION
One of the biggest challenges for all preterm infants less than 32 weeks is initiating and sustaining ventilation. Due to the functional immaturity of the lungs, this often poses a great obstacle for the very preterm infant. The development of breathing, in and ex utero, will be charted below, along with various structural properties of the lungs that lack adequate development in a newborn of low gestational age.

Development of Breathing
In utero the foetus is totally dependent on the mother for gaseous exchange. Although the foetus engages in erratic breathing-type movements, they only lead to a very small change in pulmonary volume, as foetal lungs are filled with a fluid that consists of a melange of amniotic and pulmonary fluids. It has been suggested that these movements are important for the development of the respiratory muscles, and as a means of determining the health of the foetus (Bocking and Gagnon 1991).

At birth, however a dramatic substitution takes place when the new-born has to breathe for itself for the first time. When the infant emerges from the womb, it engages in a series of gasping efforts, which increase to develop into a more regular style of breathing. After the infant stabilises, the breathing becomes quieter. It is important that the infant starts
breathing at the appropriate time. Research has shown that the actual birth plays an important role in the instigation of the breathing mechanisms. The infant is forcibly moved from a warm moist, gravitationally reduced environment, into surroundings that are much colder, have the full effects of gravity and involve the infant being handled, all of which appear to enhance post natal ventilation (Keele, Neil et al. 1983). The initiation of birth and the process of labour are also very important in helping to expel the fluid that exists in the lungs. This fluid is pulmonary in origin and acts as a plasma ultra filtrate. Some of this fluid is expelled when the infant's thorax is contracted as it passes through the birth canal. The endocrine system, which plays an important role in the initiation of labour, also helps to initiate displacement of this fluid just prior to birth (Bird, Spencer et al. 1996). Any fluid that remains is gradually removed through evaporation or drainage via the pulmonary capillaries and lymphatics.

**Role of Surfactant**

When a neonate draws its first breath of air, an air-liquid interface is formed in the alveoli. This liquid, produced by the pneumocytes, is known as surfactant (or surface active material) and is a mixture of phospholipids and proteins. Surfactant plays a very important role, especially during expiration, as it prevents the alveoli from collapsing. Alveolar pressure can be summarised according to the Law of La Place, which relates pressure (P) inside a sphere to the radius and the tension (T) in the walls. Alveolar pressure is equal to 2T/r. Pressure within large alveoli is therefore less than in the smaller ones. Smaller alveoli, however, provide a more effective means of gaseous exchange with a greater surface area to volume ratio. Without the presence of surfactant
these alveoli would collapse and no gaseous exchange could take place. Surfactant, by reducing the surface tension, decreases the muscular effort necessary to expand the lungs by a factor of 3-4. Surfactant is usually synthesised from 20 weeks onwards (Laing personal communication).

1.8 PHYSIOLOGY OF RESPIRATION
Rhythmic quiet breathing is comprised of an active (inspiration) and passive (expiration) phase. Inspiration involves an increase in thoracic capacity which is brought about by the upward and outward movement of the ribcage by the external intercostal muscles and the downward movement of the diaphragm.Expiration on the other hand causes a decrease in thoracic capacity and is brought about by the contraction of the internal intercostal muscles. During this phase the diaphragm returns to a resting position along with the ribcage. During inspiration this increase in thoracic space causes a decrease in pressure, causing air to be drawn in through the nose/mouth, past the glottis, into the trachea, down the bronchus and bronchioles to the alveolar ducts. Here the pulmonary alveoli, which are air sacs consisting of large flat epithelial cells, provide an interface for gas exchange with the pulmonary capillaries (Jennett 1989).

Gaseous Exchange
Circulation of gases in the blood is essential for survival. The arterial blood supply takes the needed inspired oxygen to the tissues for energy production, while the venous blood supply transports the carbon dioxide, one of the waste products produced by the tissues as a result of metabolism, back to the lungs for expulsion. Both the oxygen and the
carbon dioxide are loosely bound to the haemoglobin in the blood. This process of diffusion results in the arterial blood gas pressures leaving the lungs being 100mmHg for the oxygen and 40mmHg for the carbon dioxide. Temperature and carbon dioxide concentration in the blood can affect the rates of diffusion at the capillary/alveoli interface. A phenomenon known as the Bohr effect results in more oxygen being diffused if the pressure of carbon dioxide in the blood is greater, likewise if the temperature is greater than 37 degrees Celsius a similar trend is observed. Increased carbon dioxide and increased temperature are indicative of more work from the tissues and muscles, therefore requiring more oxygen and energy (Jennett 1989).

1.9 REGULATION OF BREATHING
The main aim of the respiratory control mechanisms is to ensure that some sort of pattern of breathing will be sufficient to meet the requirements of the metabolising tissues, whilst minimising the amount of muscular work required by the respiratory muscles to sustain that pattern of breathing. The respiratory regulatory system has two separate pathways that allow for voluntary as well as automatic control. Patients that are described as being in a persistent vegetative state and who have no electrical activity above the pons, can still continue with automatic breathing, with the neurones in the medulla exhibiting an inherent pacemaker-like activity.

Breathing regulation is dependent on two different feedback control mechanisms, namely mechanical and chemical. Mechanical control is concerned with the afferent information provided by chest expansion. Pulmonary stretch receptors, present on the walls of the bronchial tree,
continuously provide information about the current state of the lungs, whether they are inflating or deflating. J-receptors, on the other hand, monitor changes in interstitial pressure between the capillaries and alveoli. A combination of these different sources of information allow for modification of the depth and frequency of the breathing (Jennett 1989).

Chemical control on the other hand, uses changes in blood gas composition. Central and peripheral chemoreceptors are particularly sensitive to increases in carbon dioxide, as carbon dioxide directly affects the pH of the plasma in the body and means less oxygen is being circulated in the blood. Central chemoreceptors, based in the medulla region of the brain, detect changes in pH and bring about an increase in respiratory drive. Chemoreceptors are also located in peripheral regions of the body, namely in the aorta and carotid arteries. They can likewise detect changes in plasma pH, but also decreases in the pressure of oxygen in the blood. Such changes are quickly detected via feedback to the central respiratory centres, and adjustments in respiratory frequency and tidal volume take place (Schauff, Moffett et al. 1990).

During waking hours the body is never really in a restful state, with varying levels of activity resulting in changing carbon dioxide outputs from metabolising cells. However in spite of these disruptive processes, measurements of blood gases over the course of a day, remain remarkably constant. The heart assists by modifying the rate at which blood is circulated around the body, and therefore to the lungs, helping to expel any excess carbon dioxide.
1.10 Preterm Respiratory Problems

Premature infants, born at less than 37 weeks, may experience great difficulties breathing. Their lungs are often low on compliance, due to anatomical immaturity, and they lack the presence of surfactant. These premature infants frequently experience collapse of their alveoli which adds to the problems by significantly reducing their total lung volume. Greater respiratory efforts are also required by the infant for sufficient oxygenation, with additional ventilatory support often being needed. Bronchopulmonary dysplasia and other respiratory illnesses are resulting problems that are common in this population of infants.

Bronchopulmonary Dysplasia

Bronchopulmonary dysplasia (BPD) is a term used to describe the condition of preterm infants who have experienced some form of respiratory failure, are oxygen dependent for at least 30 days and have chronic lung pathology that is evident from chest x-rays. These x-rays usually show areas of hyperinflation, where the lung is perhaps blocked and does not deflate during expiration, and areas of atelectasis, where the alveoli in that area have collapsed, usually due to insufficient surfactant (Nickerson 1990). BPD is used to describe a wide range of chronic conditions, from those infants who have mild symptoms and are supplemented by a trickle of oxygen, to those who are ventilator dependent for several months. Between 15 and 38% of preterm infants born weighing less than 1500g and requiring mechanical ventilation, develop BPD.

The actual causes of BPD are multi-faceted, and stem mainly from the treatments administered to support the premature lungs. Infants with
BPD may have severe lung disease, with low lung compliance and poor gas exchange. It is therefore necessary to enhance airway pressure and the concentration of oxygen in the inspired air. However the toxic nature of the high oxygen concentration can result in gradual damage to the endothelial barrier and subsequent leakage of protein and fluid into the interstitial spaces and the alveoli, compromising respiration. Also the barotrauma experienced from persistent mechanical ventilation at high pressures, leads to inflammation of the surrounding tissues and further leakage of fluid into the interstitial spaces. It is difficult for clinicians to balance this trade-off between the airway pressure and the concentration of oxygen. In addition to these external factors, infection and pulmonary oedema (resulting from fluid overload or cardiac disease) can complicate the disease further (Nickerson 1990).

1.11 FEEDING PROBLEMS WITH BPD INFANTS
Infants with BPD often experience a disproportionate number of feeding problems. This is primarily due to the fact that their prematurity interferes with the normal development of the neurological and physical aspects of feeding. Being deprived of the full gestational period in utero, the preterm infant is unable to perfect the various skills necessary to feed successfully. For instance the amniotic sac provides a sub-gravitational environment that allows the various oral mechanisms to develop. It has also been suggested that the vestibular sensations provided by the mother in utero, play an important role in the development of muscle tone, tactile and proprioceptive responses (Braun and Palmer 1982). Prematurity will therefore limit the degree of uterine experience gained by the foetus.
Poor growth is also common for BPD infants, as they often need 30-50% more calories than an infant without BPD. This extra calorific intake is not just needed for the additional effort required for respiration, but also for the reparation of previous damage to the lungs. As these infants are not physically ready to feed orally until around 32-33 weeks gestational age, they receive their enriched milk quota through a nasogastric tube. Bottle feeds are usually introduced gradually after 32 weeks so the infant can become accustomed to controlling the intake of milk through sucking. Numerous problems can arise at this stage. For instance the infant may be averse to taking a teat in it's mouth, as previously it may have received a pacifier during a painful procedure, thereby making an association between oral stimuli and pain (VandenBerg and Franck 1990). Likewise intubation, insertion and presence of a nasogastric tube, and other cannula taped around the mouth, are all noxious oral experiences, which may cause the infant to be resistant to feeding. Another feeding problem associated with BPD infants is a disorganisation of the different feeding components. Some of these infants after a few weeks feeding experience can overcome this disorganisation, others may still have problems. It has been proposed that oral motor dysfunction is the cause of these persistent feeding problems, and it may be linked to neurological abnormalities. A small minority of infants examined within this group exhibited deviant jaw movements, and generalised hypotonia, suggesting that these feeding problems were indicative of cranial nerve abnormalities such as bulbar palsy or generalised central nervous system abnormalities, such as cerebral palsy (Braun and Palmer 1986).
In addition to these oral problems, BPD infants have the obvious problem with ventilation. Due to the lack of lung compliance, the extra respiratory effort required for feeding can result in respiratory muscle fatigue. Quite often BPD infants will have a significantly lower concentration of oxygen in their blood whilst feeding, a deficit they do not seem to readily overcome. This can be the result of periods of apnoea as the infant sucks. Another serious concern associated with feeding the infant with BPD is one of aspiration. A study has shown that if milk reaches the oral cavity again, through vomiting or reflux, between 60% and 80% of the infants studied aspirated (Goodwin, Graves et al. 1985). The presence of milk in the lungs can lead to further damage and infections, complicating the illness further. Extreme vigilance must therefore be exercised when feeding infants with BPD.
CHAPTER 2

MOVEMENT CONTROL

2.1 CO-ORDINATION

"The solution to the problem with co-ordination lies not in analysis of the tonal and expressive resources of a single instrument in an orchestra, but in the technical construction of the score and the mastery of the conductor."

Bernstein (1967: 91)

From the above analogy drawn by Bernstein (1967), co-ordination is more than just the production of an individual movement component (the single instrument); the desired outcome (the score) and the CNS (the conductor) have also crucial roles to play. The key to successful co-ordination can therefore be seen as the integration of lots of different movement parts to bring about a controlled approach to a desired goal. Motor co-ordination is not merely based on the "particular processes in individual neurons, but on the determinate organisation of their common activity." (Bernstein 1967)

On top of this harmonisation of the different movement parts, the CNS or the conductor, has to utilise a wealth of perceptual information received via the various sensory systems. J.J.Gibson (1966) emphasises the importance of this eclectic information in controlling actions. He reinforces the multifariousness of this perceptual information and its role in providing feedback about body movement and position and its relationship with the surrounding environment. Although vision is alleged to dominate the control of actions (Gibson 1966), the part played
by proprioception should not be underrated. Proprioception, detected by a multitude of sources, provides the CNS with valuable information about the relative position of various body parts. For instance, the mechanoreceptors in the joints, the stretch receptors in the skin and the muscle spindles in the muscles, are examples of different proprioceptive receptors (Prochazka 1996). The significance of this information, however, can most clearly be seen in its absence. Deafferented patients suffering from large fibre sensory neuropathy, experience severe impairment of limb posture and balance control (Rothwell, Traur et al. 1982), along with difficulty in regulating other voluntary movements such as reaching (Gordon, Ghilardi et al. 1995).

2.2 MOTOR CONTROL

The next question to consider, with regards to successful co-ordination, is how the CNS might integrate the perceptual information with the motor output. Descartes, in the seventeenth century, proposed that movements were controlled by sensory and motor "spirits", that flowed along different parts of the same nerve, to the CNS and the muscles respectively. In contrast Sechnov (1863) claimed that all animal and human behaviour could be reduced to simple reflex actions. He believed that sensory inflow triggered a central reflexive process, that was governed by physical laws and subsequently resulted in a motor output (as cited in Prochazka 1996).

A more recent attempt to explain motor control has focused on the idea that the brain avoids making complex computations about the forces necessary to control movements, but relies on the inherent spring-like properties of muscles instead. This equilibrium point hypothesis, or $\lambda$
model, proposed by Feldman claims that a static limb is in a constant state of equilibrium, brought about by the spinal and supraspinal systems, peripheral afferents and the spring-like properties of muscles (Feldman, Adamovich et al. 1990). A movement is instigated when the CNS initiates dynamic processes. This disrupts the previous state of equilibrium and forces the various subsystems to find a new equilibrium. The theory suggests that the CNS selects an endpoint for the movement before it begins, and then triggers a series of muscle contractions, with corresponding equilibrium points, until the destination is reached (Bizzi, Accornero et al. 1984). An arm movement could therefore be explained in terms of an equilibrium point trajectory, which would be composed of a time series of equilibrium points, sought as a result of the disruption caused by the CNS. To test this idea, Gomi and Kowato (1996) constructed a piece of apparatus to see if an equilibrium point trajectory, calculated from the theory, mapped an actual arm trajectory. When they perturbed the arm movement mid flow they found the path did not follow the trajectory predicted by the equilibrium point hypothesis. These findings indicated that the brain continued to provide input throughout the whole movement (Gomi and Kowato 1996).

2.3 TAU THEORY

Lee (1976) proposed an ecological theory that emphasised the importance of using perceptual information to control a movement prospectively. In keeping with the philosophy of movement control proffered by J.J. Gibson (1966), the tau (τ) theory stressed the need to assimilate surrounding internal and external information throughout the course of a movement, so that the approach of different body parts to a desired goal can be controlled. To illustrate this point take the simple task of bringing a
peanut to one's mouth. To be able to successfully perform this task, different sources of information, such as the distance (gap) the peanut is from the mouth and the forces that need to be applied to hold the peanut, need to be integrated by the CNS. As the action progresses, this sensory information pertaining to the movement towards the goal, will change. Co-ordination will therefore be dependent on an ability to sense how a movement, relative to a goal, is changing. With reference to the above example, the closing of the gap between the hand (the effector) and the mouth (the destination) would best be achieved if the different modes of sensory input about distance, grip force and orientation, were monitored in the same unit of measurement. Instead of having different neurally computated units of measure, the nervous system would respond to one universal measure.

Lee (1976) proposed a single measure that would provide sufficient information to control the closing of a gap. The measure, tau (τ) of the gap, is a temporal measure that corresponds to the first order estimate of the time to closure of a gap. It may be calculated by dividing a given variable x (measured in any unit e.g. cm, newtons, degrees) by the rate of change of that variable x, and may be perceived through any sensory modality. In addition to this, τ also provides sufficient information for controlling the velocity of closure of a gap. Studies have shown that by using a strategy of keeping the rate of change of τ constant, humans and even birds are able to control the velocity of their approach to a goal (Lee, Reddish et al. 1991; Yilmaz and Warren 1995). A simple time to closure measure like τ can therefore overcome the multi-unit measurement problem associated with spatial (distance and angle) and dynamic (force)
parameters, by having a $\tau$, measured in a ubiquitous unit, for each dimension.

2.4 **TAU COUPLING**

For successful co-ordination Bernstein (1967) emphasised the importance of integrating the different parts of a movement in space and time. The $\tau$ theory explains this integration in terms of $\tau$-coupling (Lee, Simmons et al. 1995). $\tau$-coupling involves keeping two different $\tau$s coupled together at a constant ratio. This integrates the $\tau$ information from the various sensory sources, so that a common goal can be achieved. Bringing the peanut to the mouth requires the utilisation of information about the distance gap $X$ and the angular gap $Y$, so that the hand can bring the peanut along an appropriate angular approach path, towards the mouth. In $\tau$ terms these two different gaps would be closed in such a way that when $\tau X$ reaches zero, $\tau Y$ also reaches zero. The most effective way of doing this would be to couple the $\tau$ of $X$ and the $\tau$ of $Y$ at a constant ratio (i.e. $\tau X / \tau Y = k$, where $k$ is a constant) throughout the movement. By coupling the $\tau$s of the gaps, the speed and the direction of the movement are synchronised so that the peanut is steered successfully towards the mouth. Lee et al (1995) have shown this to be the case for echolocating bats, when they have to control their orientation and direction when flying to collect a suspended meal worm.

2.5 **TAU - GUIDES**

Taking the idea of $\tau$-coupling further, it has been proposed that the $\tau$ of a hand movement could be coupled to another $\tau$ that is external to the body. To illustrate this concept, Figure 2.1A shows how a drummer has to move in such a way as to lock the closing of the gap ($\tau_H$) onto the conductor's
closing of the gap ($\tau_B$), so that they both reach their respective destinations at the same time.

**Figure 2.1.**
An example of a drummer using an extrinsic $\tau$-guide (A) and a corresponding intrinsic $\tau$-guide (B) when the external conductor is not available. Diagram C shows how this idea would relate to the control of sucking pressures in a newborn infant.

The conductor's $\tau_B$ acts as an extrinsic $\tau$-guide for the drummer's $\tau_H$. In
a recent experiment where subjects had to move a cursor on a computer screen to catch a moving target within a certain goal zone, the τ of the target to hand was found to act as an extrinsic τ-guide, onto which the subjects could couple the τ of the hand to goal (Lee, Clarke et al. 1997).

Bernstein (1967) proposed that the co-ordination of movement is not merely a peripheral artefact, but must emanate from the CNS as formulae or engrams of the intended movement. In the case of the drummer beating the rhythm alone (see Fig. 2.1B), it is not unreasonable to suggest that the central nervous system generates its own intrinsic τ-guides, that would provide an internal temporal representation of the movement onto which the drummer could couple the drumstick movement, to accurately produce successive strokes. Intrinsic τ-guides could therefore be viewed as an internal dynamic prototype that provides temporal information for movements to couple onto. It is hypothesised that each internal τ-guide has a single parameter, namely its duration T, and can be conceptualised in terms of an object, or energy level, that starts from rest at the same time as the drumstick starts to move towards the drum. The τ-guide accelerates (from rest) at a constant rate towards the goal place, or goal energy level, reaching it in time T (see Fig. 2.1B). (Thus at any time (t) during the movement, τ-guide (τ g) = 0.5(t-T^2/t). See Appendix I for derivations of the formula.) The drummer would therefore aim to keep the τ of the gap between the drumstick and drum (τH) coupled onto the τ-guide (τ g), in a consistent way, so that τH = k τ g, where k is a constant (see Fig. 2.1B). The value of the constant k can be set according to how the drummer wishes to approach the drum since the value of k determines the movement velocity profile. As can be seen from Figure 2.2, a k value of 0.2 produces a short acceleration to
peak velocity, with a longer more gradual deceleration phase to the target. In contrast a k value of 0.8, has a longer accelerative phase, with a shorter, more abrupt approach to the target. In general, as the value of k increases the approach to the target, namely the duration of the deceleration phase, becomes shorter.

Figure 2.2
Examples of the velocity profiles that would correspond to the different k values displayed.

![Graph](image)

Having a temporal bedrock that was \( \tau \) based would further unite the control of all movements and the perception of movements under one unit of measurement. Having a \( \tau \)-guide would therefore assist with the co-ordination of different moving components.

### 2.6 OVERVIEW OF THESIS

An internal \( \tau \)-guide is hypothesised to be the basis for the control of intraoral sucking pressures in term infants (Chapter 4). As mentioned previously sucking control is dependent on the co-ordination of the different movement components to bring about a change in intraoral pressure that will facilitate milk flow. With this being the case it is
therefore predicted that sucking control can be explained in terms of coupling the \( \tau \) of the pressure gap \( (\tau_P) \) within the mouth, where the pressure gap is the difference in pressure between the starting pressure and the desired end pressure, onto the \( \tau \) of an intrinsically generated guide \( (\tau_g) \), so that as before, \( \tau_P = k \tau_g \) (where \( k \) is a constant). Strength of coupling will be assessed by the value \( r^2 \) of the linear regression of \( \tau_P \) on \( \tau_g \) which measures the degree of linearity between the two variables.

Using the \( r^2 \) values as a measure of the strength of coupling, chapter 5 will then compare the performance of preterm infants who were born at a gestational age of less than 30 weeks with the performance of term infants. As the preterm infants are at risk of neurological impairment, it is hypothesised that significantly weaker couplings, when compared to the mean performance of twelve term infants, will be indicative of motor problems. These predictions provided by the intrinsic \( \tau \)-guide model will then be contrasted with a motor assessment given by an experienced pediatric physiotherapist when the preterm infants are at least 6 months corrected age.

As these preterm infants have known breathing difficulties, chapter 6 will investigate the effects these respiratory problems have on nutritive feeding. The frequency of sucking and breathing during burst and pause periods of feeding for preterms will again be directly compared and contrasted to the traces recorded for the term infants. Conclusions will be drawn about the effects of respiratory compromise on overall feeding performance.
3.1 BACKGROUND

In order to look at all the different facets involved when an infant feeds, it was decided to attempt to measure changes in sucking pressures, breathing rates, and gauge when a swallow occurred within the feeding cycle. It was also decided measure heart rate and concentration of oxygen in the blood, as possible additional markers of respiratory compromise during sucking. The two important considerations that had to be taken on board when selecting the most appropriate apparatus, were to limit the amount of interference with the infant's natural feeding behaviour, and to keep the cost of the equipment to a minimum.

Previous methods used by researchers to investigate sucking behaviour have included electromyography (EMG) measures from the jaw muscles (Daniels, Casaer et al. 1986), or the use of a mercury strain gauge strapped to the outside of the infant's face, again to give a measure of the movement of the jaw as the infant feeds (Bamford, Taciak et al. 1992). Both of these methods are restrictive and ignore the movement of the tongue inside the mouth. Also the use of mercury with young infants is deemed too risky by most hospital boards. Some breast feeding studies have adopted alternative ultrasonographic techniques, but the equipment is expensive and the images are difficult for novices to interpret. It was therefore decided that measuring intraoral pressure using a non-
compliant catheter protruding through a normal feeding hole, similar to the technique used by Mathew (1991), would give the most information about both jaw and tongue movements, thereby encapsulating the whole sucking action.

Different procedures for assessing breathing while feeding have included the respiratory inductance plethysmography (RIP) system, a strain gauge which monitors abdominal and chest movements (Mathew, Clark et al. 1985). Other respiratory measuring techniques include pneumotachography, a nose piece that monitors nasal airflow, or thermistors, which record temperature changes and work on the premise that inhaled air is cooler than exhaled air. In this study however, because part of the subject population would receive oxygen supplementation via nasal cannulae, measures relying on nasal airflow were discounted. Instead a specially designed system, similar to the RIP method, was considered the most appropriate to use.

In order to be able to decide where a swallow took place in the feeding sequence, a microphone seemed to be the least invasive method. Alternative techniques included monitoring changes in pharyngeal pressure, but again this involves the insertion of a cannula and could hamper the infant's normal feeding behaviour.

Pulse oximetry was found to be the most reliable and easiest way of monitoring possible fluctuations in heart rate and oxygen saturations in the blood as the infant feeds. It is a standard non-invasive procedure in most hospitals. An alternative method would have involved the use of
cardiac monitors to get measures of transcutaneous PO\textsubscript{2} and PCO\textsubscript{2}, but again this was a more intrusive method.

Ethical approval for the following methodology was granted by Lothian Health Board in June 1995. Permission was given by Royal Infirmary senior management to test in Simpson's Memorial Maternity Pavilion in Postnatal wards 52 and 53 and in the Neonatal Unit.

3.2 SUBJECTS

There were two groups of subjects. Maternal consent was a prerequisite for subject inclusion in the study.

Term

12 full term infants, 6 male and 6 female, who were at least 38 weeks gestational age at birth, took part in the study. The ages at testing ranged from 28 hours to 82 hours. 5 of these were tested again at 3 weeks of age.

Preterm

6 preterm infants were also tested. This group of infants differed considerably in gestational age (23-29 weeks) and weight (600g -1380g) when born. All infants were classified as having Bronchopulmonary Dysplasia. The criteria used for classification of BPD at the neonatal unit in Simpson's were, the infant was preterm, ventilated and oxygen dependent for at least 30 days. The preterm infants were all on nasal prong oxygen, amounts varying from a trickle (i.e. less than 0.1 litres/minute) to 0.5 litres/minute. Gestational ages when the infants had their first bottle feeds also varied (32 -37 weeks). Preterm infants
were tested once a week for four weeks, irrespective of their gestational or feeding age.

3.3 DETAILS OF FEEDING EQUIPMENT

Sucking Measure

Intraoral sucking pressures were measured using a modified Cow and Gate twist-on disposable teat, normally used in the nursery. The alteration involved the insertion of a reasonably non-compliant umbilical catheter (5 French, approx. 1.5mm in diameter), through a small incision at the base of the teat. The tube was then threaded into the inside of the teat and out through one of the three manufactured feeding holes at the top. The tube was positioned so that it was protruding about 2mm through the end of the teat. Following this modification, the teat was sterilised in a solution of Milton for at least 30 minutes. Immediately prior to testing, the teat was removed from the Milton solution and washed using sterile water.

The umbilical catheter was then connected to a 3-way tap which in turn was connected to a plastic pressure dome. The other end of the pressure dome had another 3-way tap attached (see Fig. 3.1). This disposable pressure dome fitted on top of the pressure transducer. The 3-way tap that was attached to the umbilical catheter was open to the pressure dome and the end of the tube. The second 3-way tap at the other end of the pressure dome was closed to the outside world (see Fig. 3.1). The base of the disposable pressure dome contained a membrane-like surface that moved as the pressure at the end of the catheter changed. These movements were detected by the pressure transducer, which in turn was connected to the pressure terminal on a Hewlett Packard 78342 A. The
analogue pressure readings were then converted to digital output via a DAS 800 analogue to digital conversion board in a PC 486.

To make the pressure readings more robust and less sensitive to artefactual fluctuations, air was removed from the system by priming the tube and the pressure dome with sterile water.

**Figure 3.1. A diagrammatic representation of the modified teat and the connecting pressure transducer.**

![Diagram of modified teat and pressure transducer](image)

**Pressure calibrations**

On the Hewlett Packard pressure monitor a setting of 100mmHg was equivalent to an output of 2 volts. Although the Hewlett Packard had a positive in-built calibration, a negative calibration was carried out independently as the intraoral pressures generated by the infants were going to be mainly negative. In order to do this a negative pressure generator was connected to the three way tap (where the catheter would normally be connected), and different pressures (-50mmHg to -300mmHg) produced by the generator were recorded. The positive in-
built calibration was also recorded. A graph, plotting volts recorded against mmHg generated by the HP calibrator and the negative pressure generator, was found to have an $R^2$ of 0.9999 (see Fig. 3.2). This calibration procedure was repeated at different intervals during the period of data collection.

*Figure 3.2. Calibration of positive and negative pressures.*

\[
y = 0.015482 + 0.0096605x \quad R^2 = 0.9999
\]

Heart rate and $\text{SaO}_2$

An Ohmeda 3700e pulse oximeter was used to get real time analogue output of oxygen saturation levels in the blood and heart rate.

Oximetry

The pulse oximeter operates under the premise that there are primarily two forms of haemoglobin found in the blood, namely oxygenated (with oxygen molecules loosely bound, $\text{HbO}_2$) and reduced (with no oxygen molecules attached, Hb), with both having different absorption rates for
red and infra red light (HbO₂ absorbing more infrared light and Hb absorbing more red light). The Ohmeda 3700e model overcomes the problem of distinguishing between tissue absorption and blood absorption by using a patented double wavelength pulsatile system. This involves placing the probe, which sources both red and infra red light at differing time intervals, on a pulsatile vascular area like the dorsum of the foot or a finger. The photodetector which is located on the other side of the vascular area (for example the sole of the foot or the opposite side of the finger) then monitors the modulations that are occurring as a result of the pulse, thereby eliminating false readings that would be obtained from tissue absorption. From this pulsatile reading it is also possible for the oximeter to record and display the heart rate.

In addition to the digital display at the front of the monitor, it is possible to record the real time analogue output of the saturation values and the pulse rate, by connecting two mono mini-phone output jacks to the back of the monitor. In this study the oximeter was interfaced with the DAS 800 analogue to digital conversion board, which readily accepted the 0 to 1 volt signal. A 1 volt signal represented 100% saturation of oxygen and a heart rate of 250 beats per minute. (Zero volts represented 0% saturation and 0 beats per minute). However care had to be taken to make sure the foot or hand was warm and that movement was minimised as vasoconstriction and movement can lead to false readings and zero values.

**Breathing**

Respiration traces and swallowing sounds were measured using an interface box and sensors that were specially manufactured by Densa
The respiration device, which measured changes in thoracic circumference, contained a button-like sensor held in a plastic clasp which was attached to an adjustable elasticated thoracic band (see Fig. 3.3). Inhalations (an increase in thoracic circumference) caused a tightening of the thoracic band and an inward movement of the sensor. This caused a magnet inside the sensor to move closer to a Hall strain gauge device, which produced a small current that was amplified to give a respiration signal. Exhalation (a decrease in thoracic circumference) caused the band to slacken, thus decreasing the pressure on the sensor and decreasing the current. The analogue output was in wave form. The baseline was 2 volts and the range was 0.6 to 3.8 volts. Studies at Great Ormond Street showed that the timing measures given by this Densa system were in close agreement with those obtained using respiratory inductance plethysmography (RIP), another method of assessing respiratory function that looks at changes in the cross sectional area of the thorax (Ross Russell and Helms 1994).

**Figure 3.3** The thoracic band designed by Densa to measure changes in respiration.
Swallowing
Swallows were recorded using a custom-made microphone. The microphone had a thick rubber cover to eliminate as much external noise as possible and an internal filter to exclude frequencies greater than 800 Hz. Analogue output from the Densa box was on a 1 volt to 2 volt log scale.

Initial trials were carried out to see how swallowing recordings taken from the microphone compared with those heard through a stethoscope. An experienced midwife listened with a stethoscope and triggered a button every time a swallow was heard. Analysis showed a reasonable relationship between the trace from the microphone and the trigger from the stethoscope. Swallows appeared to be characterised by sharp spikes increasing suddenly from the base level. However when it came to analysing the microphone traces during a feeding episode, the swallows were much more ambiguous and difficult to reliably detect. This lack of reliability prevented an in depth analysis of the microphone recordings.

Analogue to Digital Conversion
The five analogue channels, namely pressure, sound breathing, oxygen saturations and heart rate, were all interfaced and converted to digital output using a DAS 800 analogue to digital board. The DAS 800 A/D card had an output in the range of -5 to +5 volts. The board was configured for a PC 486 DX.

The software for data collection was programmed in Visual Basic and executed in Windows 3.1. Two different programmes were written, one sampling all five channels, fifty times a second and another sampling
only the pressure channel at 200 times a second. Limitations with the software meant that the maximum number of samples was 32000. This meant a sampling rate of 50 Hz for each of the five channels, which restricted data acquisition time to 128 seconds. For the single pressure channel, a sampling rate of 200 Hz limited recording time to 39.9 seconds.

3.4 PROCEDURE

For the full term infants mothers were approached on the postnatal wards and given details of the study. Following informed maternal consent, the mother brought her baby to the postnatal nursery when the baby was alert and demanding its next feed. In the case of the preterm infants, the baby stayed where its cot was positioned within the neonatal unit. The mother sat in one of the nursery chairs with the infant on her knee. Before the mother lifted the infant from the cot, the thoracic band, with the plastic buckle attached, was positioned by the experimenter on the infant, over a close fitting babygro. Using the velcro parts of the straps the band was adjusted so that it was mid abdomen between the navel and rib cage with the plastic buckle being held in a fixed central position. The small button-like sensor was then placed inside the plastic buckle, next to the babygro. The microphone was positioned slightly to one side of the infant's oesophagus in the mid neck region and was secured using some lightly sticky micropore tape. If the infant's foot was not bare, the necessary clothing was removed, so that the pulse oximeter probe could be attached. The probe had two circular parts about 3 cm apart, one for emitting red and infra red light and the other, a photodetector, for monitoring the modulations in the light as it passed through the infant's blood and tissues. One part of the probe was placed
on top of the foot, the other placed on the bottom, with the position being secured using a band of soft material with velcro ends. When the pulse oximeter was attached, all equipment was switched on and connections were checked. The pulse oximeter took a couple of minutes to pick up a signal. Once a reading was established the fast response option was selected on the front of the monitor. This fast response provided an output of a weighted average of the oxygen saturation and pulse rate readings every 3 seconds. A standard bottle of milk (either Cow and Gate or SMA Gold Cap) normally used in the nursery was attached to the modified disposable teat. The Hewlett Packard pressure monitor was zeroed and calibrated using the in-built calibration. The necessary application program to collect the data was opened in file manager in Windows. A new folder was created for each subject and a new file for each recording for that subject. When everything was ready the mother was given the bottle. The pressure dome was held by the experimenter marginally above the bottle to prevent back flow of milk into the sterile water in the pressure dome. As soon as the bottle entered the baby's mouth, the recording was started. The pressure changes were observed on the screen of the Hewlett Packard monitor, and the oxygen saturation and pulse rate on the digital display given by the pulse oximeter. The mother was encouraged to feed the baby as normal. The limitations of the software meant that the recording was automatically stopped after 128 seconds. After this time the output from the various channels was displayed on the computer screen. A new file was opened and recording started as soon as possible. The mother was encouraged to wind the baby at these points, but often the mother felt that the infant was not ready. It also was not desirable to continuously disrupt the infant's natural rhythm. If, however, winding was required mid recording, data collection
was stopped at that point, and restarted when the infant was ready to start feeding again. All files were saved in an ASCII format.

Any part of the apparatus that came into direct contact with the infant was subsequently discarded and a new set-up was prepared for the next session. The dome and 3-way taps were sterilised daily and then replaced after a week's usage. Five of the term infants were tested as close to three weeks of age as possible, and five of the preterm infants were tested four times, with the other one being tested three times. Details of ages at testing are given in the results section.
CHAPTER 4

SUCKING CONTROL IN TERM INFANTS

4.1 SUCKING PRESSURES

The intraoral pressure changes that were measured inside an infant's mouth while bottle feeding were examined in closer detail. The pressure traces recorded were cyclical in nature, with sucks being characterised by an increase in negative pressure (suction), followed by a subsequent decrease in negative pressure (suction) (see Fig. 4.1).

Figure 4.1
Sucking pressures and the corresponding velocities, recorded from a 2 day old term infant during bottle feeding.

This pattern appeared to mirror the two distinct movement processes that are required for successful sucking. When the corresponding velocities (i.e. rates of increase) were calculated for the sucking pressure
traces using finite differences, it was noticed that the velocity profile for a suck consisted of two different turning points: a trough during the increasing suction phase and a peak during the decreasing suction phase (see Fig. 4.2).

Figure 4.2
A diagrammatic representation of the distinct movement components of a suck and the corresponding pressure trace and velocity profile.

Interestingly both the velocity profiles for the increasing suction phase and the decreasing suction phase were bell shaped, with an acceleration and a deceleration phase (see Fig.4.2), resembling velocity traces for a
typical reaching movement (Jeannerod 1988). This is quite remarkable given the different synergies of muscles that have to be co-ordinated to produce such pressure changes in the mouth and the joint articulations involved in reaching.

The above pressure trace in Fig. 4.2 differs markedly from the recording of the increasing and decreasing suction pressures registered using a mechanical breast pump (see Fig. 4.3 below). In the pump, the suction is increased to a maximum and then released to allow the nipple to refill. As can be seen the increasing suction phase is significantly longer than the decreasing suction phase with the characteristic smooth bell shaped profile observed in a term suck cycle being absent.

Figure 4.3
The pressure trace and corresponding pressure velocity recorded from a breast pump.

4.2 CONTROL OF SUCKING
It is important for the feeding infant to be able to precisely control the pressure changes inside the mouth, to maximise the flow of milk from the
bottle, and also to carefully move the expressed milk to the back of the mouth, ready to be swallowed. In order for the infant to be able to control these intraoral pressure changes, there must be some internal mechanism monitoring these changes. Pacinian corpuscles found deep in the skin are known to provide the nervous system with sensory feedback about the changes of pressure exerted on the surface of the skin (Schauff, Moffett et al. 1990). With reference to sensing intraoral pressure changes, experiments using microneurography to record the electrical activity from single nerve fibres, have shown that afferents in the oral mucosa innervated by the infraorbital nerve, increased their firing rate when the intraoral pressure changed as a result of the subject blowing (Furusawa, Yamaoka et al. 1992). Furusawa, Yamaoka et al. (1994) extended this research to look at the implications of intraoral pressure build-up during phonation. The data suggested that the significant difference in the responsiveness of the receptors supplying the oral mucosa during /pa/ production, when compared to /ta/ and /ka/ production, was linked to the different intraoral pressures required to produce those sounds. Our ability to sense intraoral pressure is therefore very important for communicating and producing different speech sounds.

As intraoral pressure changes when sucking are the end result of the coordination of different moving parts (see Fig. 4.2), control of sucking, as mentioned previously, is in many ways analogous to the control of reaching. Reaching movements are characterised by a bell shaped velocity profile, indicating an acceleration and a deceleration phase which can be modulated according to the subject's specification (Jeannerod 1988). Pressure traces recorded during sucking show a similar pattern
(see Fig. 4.2). Co-ordinating these pressure changes and controlling the movement of other body parts, may therefore involve the recruitment of the same underlying principles of movement control. One of these principles may be the existence of intrinsic $\tau$-guides, that provide temporal information for the $\tau$s of movements to couple onto, at a constant rate (see Chapter 2 for a theoretical overview). To test the application of an intrinsic $\tau$-guide model to explain the control of sucking, analysis was carried out using the following procedure.

4.3 **CALCULATING AN INTRINSIC $\tau$-GUIDE**

Manipulations on the data were carried out in Kaleidagraph, a graphing and data analysis package for the Macintosh, using the formula bar and pre-programmed macros (see Appendix I for formulae).

**Suck Selection**

Sucks for analysis were selected from the 200 Hz pressure recordings. In order to reduce the effects of noise all of the recordings had previously been smoothed using a Gaussian filter with time constant sigma of 20ms. Sucks registering a pressure of less than -25 mmHg were deemed not to be a proper suck, and were excluded from the analysis. Likewise any sucks that were markedly different from the surrounding pattern were also discounted. In order to get a good representation of sucks from the entire feeding session, all sucks meeting the above criteria were numbered. A random numbers sequence generated by the computer, was then used to select 12 sucks by each infant for detailed analysis.

**$\tau$-Guide Analysis**

The following manipulations were performed on the individual sucks. Each suck was divided into the increasing suction phase and the
decreasing suction phase. The increasing suction phase was considered to be the period during which the pressure trace progressed from the least negative point (peak) to the most negative point (the subsequent trough). The decreasing suction phase followed on from the increasing suction phase, and was taken from the trough where the increasing suction phase ended to the next immediate peak (see Fig. 4.2).

Each phase of the suck was treated separately in the analysis as it was characterised by a different movement. For each phase a time series was created, running from zero at the start of the phase, in increments of 0.005 seconds (the sample interval), to the end of the phase. Times for a phase varied from 0.15 seconds to 0.6 seconds. The pressure data were then normalised by adding or subtracting (whichever was appropriate for the increasing suction phase or decreasing suction phase), the last value in the pressure data set from each of the other pressure values in that data set. In the case of the increasing suction phase the normalised pressure values were positive going to zero, and for the decreasing suction phase, the normalised pressure values were negative going to zero. The rate of change of the normalised pressure at each sample time \( t \) was then calculated using finite differences. Finally the \( \tau \) of normalised pressure \( (\tau_p(t)) \) at each sample time \( t \) was calculated by dividing the normalised pressure values \( (p(t)) \) at time \( t \) by the rate of change of the normalised pressure \( (\dot{p}(t)) \). That is:

\[
\tau_p(t) = \frac{p(t)}{\dot{p}(t)}
\]

For each phase the guide \( \tau \) \( (\tau_g(t)) \) at each sample time \( t \) was then calculated using the formula:-
where $T = \text{duration of the sucking phase}$

(See Appendix I for derivations of the formula.)

The hypothesis is that, during each phase of the suck,

$$\tau_p(t) = k \tau_g(t)$$

for some value $k$ that is constant throughout the phase. The best fit value of $k$ was determined by the following procedure. First, in order to eradicate very large $\tau$ values that were a consequence of small velocities at the very start and end of the movement, those values that fell below 10% of the peak pressure velocity during the phase, were removed. (This was usually only the first and last couple of data values). The best-fit guide was then found using the least sum of squares criterion to calculate the constant $k$. Following this the strength of coupling between the $\tau$ pressure and the $\tau$-guide was measured by calculating the $r^2$ value a means of assessing the degree of linearity between the two variables (see Appendix I for equations). The calculated $k$ and resulting $r^2$ values for each phase of the suck are discussed below.

### 4.4 TERM RESULTS

All twelve term infants tested were born at a gestational age of 38 weeks or greater, with the post natal age at the time of testing varying from 28 to 82 hours. The resulting $r^2$ and $k$ values from the $\tau$-guide analysis are discussed separately.

**$r^2$ values**

As can be seen from Figure 4.4, the mean $r^2$ values of the 12 sucks for each individual infant, for the increasing suction and decreasing suction
phases, are all above 0.95 (apart from one extraneous value), indicating a strong coupling between the $\tau$ of the pressure and the $\tau$ of the guide.

**Figure 4.4**
Mean $r^2$ values for the increasing and decreasing suction phases for 12 term subjects. The bar chart on the left shows the actual means obtained by each subject in each phase, whereas the boxplot on the right shows the spread of all the subjects' means for the two different phases.

Looking at the results for the different phases in more detail, the box plot shows that the mean $r^2$ values for the increasing suction phase, range from 0.955 to 0.996, with a median value of 0.982 (indicated by the middle thick black line). The overall mean for all 12 infants for this phase was found to be 0.981, with a standard deviation of 0.01. The decreasing suction phase, however, had a wider spread of mean $r^2$ values, ranging from 0.901 to 0.992 (see Fig. 4.4). The median value in this case was lower than the increasing suction phase at 0.975. Again the overall mean for all 12 infants was lower at 0.967, and the respective standard deviation was higher at 0.025. It might be worth noting that the outlier values (represented by a circle) in both boxplots corresponded to the same infant (subject number 3).

**k values**

From Figure 4.5 below, the bar chart and the boxplot both show that there is something very different about the approach to the destination
for the increasing suction phase, compared to the decreasing suction phase. The mean k values for the increasing suction phase vary quite considerably from 0.427 to 1.13, whereas the mean k values for the decreasing suction phase range from 0.11 to 0.42.

Figure 4.5
Mean k values for the increasing and decreasing suction phases for 12 term subjects. The bar chart on the left shows the actual means obtained by each subject in each phase, whereas the boxplot on the right shows the spread of all the subjects' means for the two different phases.

However it should be noted that when the $r^2$ is low the k value loses its reliability as an indicator of how the movement is controlled. If $r^2$ is high (around 0.95) then the k values provide information about how the movement is approaching its destination. A k value ranging from 0 to 0.4 indicates that peak velocity was reached during the first half of the movement time with a gentle deceleration to the destination. A value of greater than 0.4 but less than 1 demonstrates a more abrupt approach to the destination, suggesting that peak velocity was reached in the second half of the movement (see Fig. 2.2 for examples). For the increasing suction phase, the infant appears to adopt an approach to the peak negative pressure that is more sudden (mean k value 0.68), whereas a more gradual approach (mean k value 0.2) is used during the decreasing suction phase. The difference between the two phases was found to be statistically very significant ($t_{(11)}=6.39; p=0.0001$).
In order to see how consistently the infants used an intrinsically generated τ-guide to control sucking, as opposed to an intrinsically generated regular temporal rhythm, coefficients of variation were calculated for the $r^2$ values (which measure the strength of coupling), the movement times and the changes in pressure that occurred, for both the increasing and the decreasing suction phases (see Fig. 4.6).

**Figure 4.6**
The two graphs illustrate the coefficients of variation calculated from the movement time taken, the changes in pressure that occurred, and the $r^2$ values showing the strength of coupling between the τ-pressure and the τ-guide; for both the increasing and the decreasing suction phases.

The coefficient of variation was calculated by dividing the standard deviation of a given parameter, by the mean of that given parameter. A
high coefficient of variation indicates a high degree of variation relative to the mean, and therefore a lack of consistency within that measure.

From Figure 4.6 it can be seen that all the subjects (apart from subject 3 in the decreasing suction phase) have strikingly higher coefficients of variation for both the movement time and the changes in pressure when compared to the r² values. This noticeable difference would suggest that keeping the sucking times and pressures constant is not of prime concern to the infant when controlling a series of sucks.

It would appear that it is much more important for infants to be able to control sucking using an internal mechanism, such as the τ-guide, that allows for variability in the movement times and the pressures generated when sucking, to accommodate changing environmental demands, like increased milk flow (Mathew 1991).

4.5 DISCUSSION

It would therefore appear that the combination of the jaw, tongue, lip and buccal movements, while bottle feeding, are integrated in such a way that they bring about smooth pressure changes within a newborn infant's mouth. The evidence for adherence to an internal temporal leader (the intrinsic τ-guide) is supported by the results. The high mean r² values obtained for the 12 newborn infants, suggest the existence of a strong coupling between the τ of the pressure and the τ of the proposed constant-acceleration-from-zero-velocity guide. This pattern was evident for both the increasing and the decreasing suction phases. The decreasing suction phase did show more variation and did have mean r² values that were significantly less than the mean r² values for the increasing suction
phase \((t_{11}=2.15; \ p=0.055)\). This finding could be explained by examining more closely the purpose of the decreasing suction phase of a suck. The infant is aiming to propel the milk to the back of the mouth in a controlled way, but the pressure changes recorded may be influenced by the dynamic properties of the moving milk. This might make it harder to couple perfectly the \(\tau\) pressure with the internal guide.

The mean \(k\) values for each phase also provide interesting information about how the pressure changes in the mouth are controlled. The two different suction phases serve two very different purposes in the suck cycle. The increasing suction phase is concerned with optimising the flow of milk from the feeding vessel into the mouth. This purpose is reflected in the mean \(k\) values that are significantly greater than 0.4 \((t_{10}=4.82; \ p=0.0007)\), highlighting a longer period of acceleration and a more abrupt approach to the destination (see sample \(k\) velocity curves in Fig. 2.2). The opposite was found for the decreasing suction phase, where a significantly gentle approach was found when the mean \(k\) values were compared to 0.4 \((t_{10}=-7.43; \ p=0.0001)\). This strategy is definitely more advantageous for the infant, as the danger of expressed milk coming into contact with the infant's airway, is minimised. Instead, the milk is collected in a controlled way at the back of the mouth where it awaits swallowing.
CHAPTER 5

SUCCING CONTROL IN PRETERM INFANTS

5.1 PRETERM FEEDING

Infants born at a very low gestational age (less than 30 weeks) are usually able to suck and swallow well enough to gain adequate nutrients around the 32nd week of gestation (Hack, Estabrook et al. 1985). Successful sucking and swallowing is dependent on intact brain stem pathways and the transmission of impulses through the cranial nerves to healthy musculature in the mouth, tongue and pharynx. Injury to, or dysfunction in any of these regulatory systems could manifest itself in abnormal sucking behaviour (Coulter and Danner 1987).

Figure 5.1
Examples of the distance and velocity traces of adults bringing their hand to their mouth.

In the field of motor control, the literature points to the finding that neurological insult is characterised by a lack of control that is evident
from the erratic positional and velocity traces observed when performing a movement (Isenberg and Conrad 1994). This is clearly illustrated in the case of Parkinson's Disease, cerebral palsy and patients suffering from lesions to the cerebellum. Some data collected in the perceptuo-motor laboratory illustrates this point quite well by showing an obvious difference between a normal adult, and an adult patient with a cerebellar lesion, bringing their hands to their mouths to eat a grape (see Fig. 5.1). The velocity profile elicited by the cerebellar patient shows marked perturbations, when compared to the smooth trace of the other adult.

The intraoral sucking pressure and corresponding sucking pressure velocity traces may therefore provide an indication of the smoothness and skill of the oral motor control.

Figure 5.2. An example of the sucking pressure traces and corresponding pressure velocities recorded from a 39 week old preterm infant (born at 23 weeks gestational age.)
Perhaps, therefore, preterm infants born at a very low gestational age and who are at-risk of incurring a neurological insult, may show some abnormalities in the sucking pressure velocity traces (see Fig. 5.2).

As all twelve of the healthy term infants tested have shown a strong coupling between the $\tau$-pressure and the intrinsic $\tau$-guide in the previous chapter, abnormalities in the preterm performance may be exposed by a significantly weaker coupling.

5.2 PRETERM RESULTS

Due to the nature of their current clinical conditions and the varying gestational ages at birth, the six preterm infants tested were not seen as a homogenous group, and were treated as individual cases. For each preterm a brief clinical history will be given along with any specific caregivers comments on feeding performance at the time of testing. Sucks for the guide analysis were selected in exactly the same manner as for the term infants, with the same method for calculating the intrinsic $\tau$-guide being used for each suck (see Chapter 4.3).

Preterm P1

Preterm P1 was born at 23 weeks gestational age, at a weight of 0.645 kg. At the time of the first test the infant was 35 weeks gestational age and had been receiving a bottle a day for around five days. (The other feeds were nasogastric tubes). She was receiving 0.1 litres of oxygen per minute supplementation through nasal prongs. Figure 5.3 shows her progress in both the increasing suction phase and decreasing suction phase components of the suck cycle, as she was monitored weekly over four consecutive weeks. Her mean $r^2$ for the increasing suction phase never overlapped with the term range, with her performance in the
fourth week of testing declining markedly. The decreasing suction phase was considerably better with weeks 1, 2 and 3 all falling within the term range. However week four again showed a significant fall in previous performance levels. The mean fell to 0.843 and the standard deviation increased to 0.186. It might be worth noting that at this stage in the infant’s development the caregivers remarked that they had noticed the infant engaging in tongue thrusting.

**Preterm P2**

Preterm P2 was a twin born at 24 weeks gestation, with a birthweight of 0.665kg. He was being supplemented with < 0.1 litres of oxygen per minute, and was 33 weeks gestation at the time of the first test session. As can be seen from Figure 5.3., his r² values for the increasing suction phase fall within the term range by the third week of testing (weeks 1 and 2 are well out of these limits). By the fourth week (36 weeks gestational age), these values have increased so that they are verging on the mean value achieved by the newborn terms (0.98). Mean k values for weeks 3 and 4, are 0.68 and 0.41 respectively. The decreasing suction phase values however, consistently fall within the term range across all the testing sessions, with the final session having a mean r² of 0.99. Mean k values range from 0.09 in the first session to 0.38 in the final session, indicating a more moderate approach to the destination point, like the terms.

**Preterm P3**

Preterm P2's twin was 0.6 kg when born. He did not start bottle feeding until 3 weeks after his brother, as he suffered a serious setback when he had to undergo laser treatment for retinopathy of prematurity. When he
did commence feeding at 36 weeks gestational age, he consistently fell within the limits set by the newborn terms for both sucking phases, with the decreasing suction phase values even being above the mean term values at each testing session (see Fig. 5.3).

Figure 5.3. The mean $r^2$ values for preterms P1, P2 and P3 for the increasing suction phase (red graphs) and the decreasing suction phase (blue graphs). The term range is defined by the dotted box, with the mean being shown as a line within this box.
Mean k values for the increasing suction phase ranged between 0.5 and 0.8, with the decreasing suction phase k values spanning from 0.18 to 0.3. For the first two weeks of testing Preterm P3 was supplemented with <0.1 litres of oxygen per minute. This was increased to 0.2 litres of oxygen per minute, following a clinical assessment, for the third and fourth testing sessions.

**Preterm P4**
Born at 25 weeks, weighing 0.775 kg, preterm P4 was not able to suck for a sufficient length of time in the first testing session to collect some 200Hz pressure data. She was 37 weeks at the time of the first test and she was being supplemented with 0.2 litres of oxygen per minute. During the following 3 weeks, she was able to suck for longer periods of time, but the analysis showed that her r^2 fell well below the term range for the increasing suction phase. However the decreasing suction phase showed improvement, with the final session indicating r^2 results that were on a par with the term findings (see Fig. 5.4). Again k means were between 0.17 and 0.39. For recordings 3 and 4, preterm P4 was receiving 0.5 litres of oxygen per minute.

**Preterm P5**
Preterm P5 was 1.38kg when she was born at 28 weeks gestation. She began bottle feeding at 36 weeks, when she was being supplemented with < 0.1 litres of oxygen per minute. She showed consistent improvement in her r^2 values for the increasing suction phase, with the fourth testing being in the lower end of the term spectrum (see Fig. 5.4). The decreasing suction phase consistently reached the term standard, with weeks 3 and 4 being above the newborn term mean. K values for the
decreasing suction phase were quite varied across the 4 weeks, ranging from 0.08 to 0.4. The increasing suction phase k values for weeks 2, 3 and 4, ranged from 0.69 to 0.41.

Figure 5.4 The mean $r^2$ values for preterms P4, P5 and P6 for the increasing suction phase (red graphs) and the decreasing suction phase (blue graphs). The term range is defined by the dotted box, with the mean being shown as the line within this box.
Preterm P6

Preterm P6 was the oldest at birth (29 weeks), weighing 1.34 kg. He was heavily ventilated for the first two weeks of his life, and did not start bottle feeding until 40 weeks. He received 0.35 litres of oxygen per minute at the time of the first test, and 0.5 litres of oxygen per minute for the other tests. A fourth test was not possible as he stopped feeding after the third test, and never bottle fed again. All his increasing suction phase $r^2$ values across the three different sessions were notably lower than the term standard (see Fig. 5.4). The decreasing suction phase values did just fall within the term range in the first two sessions, but fell away again in the third and final session.

5.3 SUMMARY OF Z-SCORES

Each individual preterm's mean $r^2$ scores were converted to standard scores (z-scores), to see if their performance in each phase of sucking fell within the boundaries of the term population.

Z scores were calculated using the standard formula:

$$z = (\text{preterm mean } (X) - \text{term mean } (\mu)) / \text{term stdev } (\sigma)$$

with the significance of the resulting z-scores being checked against a standard z-score table (areas under a normal distribution). Below are the tables summarising the results for the increasing suction phase and the decreasing suction phase.

As can be seen from Table 5.1, at no time over the four testing sessions did Preterm P3 differ significantly from the term means for the increasing suction phase. Only preterm P2 (P3's twin) reached the same standard by the 4th testing session, falling statistically within the term distribution.
Table 5.1.
Summary of significance of z-scores for mean $r^2$ values for the increasing suction phase for preterm infants.

<table>
<thead>
<tr>
<th>Test Session</th>
<th>1st</th>
<th>2nd</th>
<th>3rd</th>
<th>4th</th>
</tr>
</thead>
<tbody>
<tr>
<td>P1</td>
<td>-14.8 ***</td>
<td>-24.42 ***</td>
<td>-4.41 ***</td>
<td>-10.18 ***</td>
</tr>
<tr>
<td>P2</td>
<td>-8.97 ***</td>
<td>-13.29 ***</td>
<td>-1.64 *</td>
<td>0.09</td>
</tr>
<tr>
<td>P3</td>
<td>0.90</td>
<td>0.71</td>
<td>0.55</td>
<td>1.3</td>
</tr>
<tr>
<td>P4</td>
<td>no test</td>
<td>-8.32 ***</td>
<td>-12.03 ***</td>
<td>-6.87 ***</td>
</tr>
<tr>
<td>P5</td>
<td>-19.98 ***</td>
<td>-7.12 ***</td>
<td>-2.89 **</td>
<td>-1.98 *</td>
</tr>
<tr>
<td>P6</td>
<td>-6.87 ***</td>
<td>-12.91 ***</td>
<td>-14.36 ***</td>
<td>no test</td>
</tr>
</tbody>
</table>

* p<0.05
** p<0.01
*** p<0.001

Preterm P5 showed some improvement over the four testing sessions but still managed to fall significantly below the term means. By the fourth session preterms P1 and P4 were still very significantly different from the term means, while P6 had stopped feeding altogether.

Table 5.2.
Summary of significance of z-scores for mean $r^2$ values for the decreasing suction phase for preterm infants.

<table>
<thead>
<tr>
<th>Test Session</th>
<th>1st</th>
<th>2nd</th>
<th>3rd</th>
<th>4th</th>
</tr>
</thead>
<tbody>
<tr>
<td>P1</td>
<td>0.92</td>
<td>-2.17 *</td>
<td>0.76</td>
<td>-5.03 ***</td>
</tr>
<tr>
<td>P2</td>
<td>0.27</td>
<td>-0.71</td>
<td>0.32</td>
<td>1.01</td>
</tr>
<tr>
<td>P3</td>
<td>0.96</td>
<td>1.03</td>
<td>0.92</td>
<td>0.6</td>
</tr>
<tr>
<td>P4</td>
<td>no test</td>
<td>-3.87 ***</td>
<td>-1.94 *</td>
<td>-0.19</td>
</tr>
<tr>
<td>P5</td>
<td>0.36</td>
<td>0.03</td>
<td>0.89</td>
<td>0.42</td>
</tr>
<tr>
<td>P6</td>
<td>-1.84 *</td>
<td>-1.77 *</td>
<td>-5.28 ***</td>
<td>no test</td>
</tr>
</tbody>
</table>

* p<0.05
** p<0.01
*** p<0.001

Table 5.2 shows an altogether different picture for the decreasing suction phase. All apart from preterm P1 had reached the term standard for the decreasing suction phase by the 4th week of testing, with some of the
preterms falling within the term range from the 1st week of testing (P2, P3 and P5).

This obvious difference between preterm performance in the increasing and the decreasing suction phases may partly be attributed to the smaller mean and larger standard deviation for the term decreasing suction phase (mean=0.966, stdev=0.025), compared to the term increasing suction phase (mean=0.981, stdev=0.01).

5.4 MOTOR DEVELOPMENT ASSESSMENT
In order to appraise the possible relevance of the preterm sucking control data to predict future motor outcome, a motor development assessment was carried out by a physiotherapist specialising in paediatric care. The physiotherapist was unaware of the outcome of the sucking control data. Three out of the six preterm infants (corrected age greater than 6 months) were videoed, and subsequently assessed. (Preterms P1, P2 and P3 were unfortunately not old enough to undergo this assessment at the time of thesis submission.) The physiotherapist’s appraisal consisted of three different categories. The first involved looking at the infant’s response to sudden changes in posture, as determined by the Vojta Scale (see Appendix II for the notes and different postures used). The physiotherapist would assess how many of these postural reactions were presented normally. The second area was a combination of motor developmental milestones reached and the physiotherapist’s assessment of gross and fine motor control. Gross and fine motor function were judged by the infant’s ability to reach and grasp, along with general self-propulsion movements on the floor. The third and final category involved
general observations about the infant's degree of muscle tone. The physiotherapist's summary of each infant's performance will be given below.

**Preterm P4**

At the time of assessment P4 was 9 months corrected age (1 year and 3 weeks old, minus 15 weeks to correct age). All the postural reactions on the Vojta Scale were presented normally, within the 9 to 12 month movement quality range. Her gross motor functions, such as the transition from free sitting to crawling, were again within the expected age range. However her fine motor function, namely the scissors grasp, was slightly delayed (8 month level). The physiotherapist also noted that P4 exhibited a mild degree of hypotonia in classic areas seen in preterm infants, particularly the shoulder girdle, abdominal muscles and the hip adductors. This meant that the quality of her gross motor function lacked stability and smoothness, which the physiotherapist attributed to poor muscular power. In spite of this, however, the physiotherapist concluded that on the whole her a functional motor performance with respect to the movements she could perform, was more or less within a normal range.

P4's sucking performance in the increasing suction phase, according to the t-guide model, had improved by the fourth session (P4 was 41 weeks gestational age), but was still very significantly different from the term standard (p<0.001). Her decreasing suction phase likewise improved across the test sessions, so that by the final session it was no longer significantly different from the term mean.
Preterm P5

P5 was 6 months at the time of assessment (9 months minus 13 weeks to correct age). Four out of the 7 postural reactions were classified as normal. In the other three she tended to stiffen her legs and clench her fist, which according to the physiotherapist, indicated some irritability of the CNS. The physiotherapist also reported a 2 month delay with her gross motor functions, particularly her reaching and grasping ability, but did not make any reference to observed hypotonia. She did mention a lack of stability in the abdominal area.

P5's sucking control results for the increasing suction phase improved dramatically from the first session (36 weeks gestational age with a mean $r^2 = 0.78$) to the final session (39 weeks gestational age mean $r^2 = 0.96$), but still fell outwith the term standard ($p<0.05$). However, the decreasing suction phase was always within the term mean boundaries, for all of the sessions.

Preterm P6

P6 was 7 months corrected age (9.5 months minus 11 weeks to correct for prematurity). Five out of seven of the postural changes were "normal" at the 6 month level, when responding to sudden changes in posture. The other two responses showed marked immaturity. Gross motor activity was classified as partly age appropriate (e.g. rolling over back to front), but in other areas function was dramatically delayed exhibiting a 2 month old level of ability. The physiotherapist concluded that P6 suffered from a moderate degree of hypotonia that impaired his development of gross motor function. He had poor muscular power in all areas, but particularly in the trunk region. He was unable to integrate
the various muscle groups to pull himself up, therefore limiting his chances of interacting with the surrounding environment, and enhancing his proprioceptive and sensory experiences to prepare for other gross motor activities.

P6's sucking performance in both the increasing suction and decreasing suction phases was very poor. In fact P6 developed a feeding aversion after the third testing session, and refused bottle feeds thereafter.

5.5 CONCLUSIONS

In the increasing suction phase all the preterm infants, apart from P3, were found to have significantly weaker couplings than the term infants between the $\tau$ pressure and the $\tau$-guide. However, by the fourth week of feeding experience most of the preterm infants had improved the strength of the $\tau$-coupling. In contrast the decreasing suction phase did not show such marked differences between the preterm and term infants. Four out of six of the preterm infants fell within an acceptable limit of the term range. As has been mentioned earlier, a possible reason for this discrepancy may be the wider range of mean $r^2$ recorded for the term infants. An alternative explanation may be that the control or the need to couple the $\tau$ of the intraoral pressure may not be as essential in this phase.

Certain aspects of the motor assessment were in accordance with the sucking data for preterms P4 and P5. For instance, P5 was still significantly below the term mean $r^2$ values for the increasing suction phase at 39 weeks gestation and subsequently showed a 2 month delay in her motor performance when assessed by the physiotherapist at 6
months corrected age. P4 likewise was significantly below the term mean $r^2$ values for the increasing suction phase at 41 weeks gestation. She showed a one month delay in her fine motor control and was found to lack stability and smoothness in her gross motor function. Neither of these preterms were classified as having age appropriate motor development. The $τ$-guide sucking analysis did therefore appear to flag early differences in the motor performance of these preterm infants when compared to term infants. More detailed assessments would be required to draw more precise conclusions about the connection between sucking control and later movement control.

For P6, however, there does appear to be a much more definite association between the poor sucking performance and his subsequent impaired motor performance at 7 months corrected age. All facets of his motor appraisal were flawed in some way. Particularly noticeable was the degree of hypotonia that impeded further development of function. The root of this hypotonia is most probably neurological.
6.1 INTRODUCTION

A very important aspect of feeding is the maintenance of an adequate level of ventilation while the infant engages in sucking and swallowing. How and to what extent ventilation is compromised is still not very well understood. It has been reported that term infants, less than 48 hours postnatal age, significantly reduced their breathing rate and tidal volume whilst sucking (Bamford, Taciak et al. 1992). A similar pattern was also observed in preterm infants when comparing ventilation in the sucking periods to that in the pause periods (Shivpuri, Martin et al. 1983). However as preterm infants are prone to episodes of apnoea when feeding (Guilleminault and Coons 1984), a lack of cardiorespiratory maturity might be implicated in the inability to co-ordinate respiration effectively (Bamford, Taciak et al. 1992). It has been suggested that with increasing development a better suck:swallow combination emerges, resulting in less disruption to the breathing pattern (Shivpuri, Martin et al. 1983).

Two distinct patterns of feeding have been identified in the newborn infant, namely continuous and intermittent, and are thought to be important in the regulation of ventilation (Mathew 1991). The continuous phase occurs at the start of a feed when the infant sucks non-stop for at least 30 seconds, usually longer. During this time airflow is constantly interrupted by the need for regular swallowing. Following this stage the infant sucks more intermittently, with short sucking bursts being followed by pauses. Mathew (1985) proposes that these pause
periods in this intermittent feeding stage allows the infant to restore previous levels of ventilation. However as oxygen saturations have been found to remain relatively constant throughout a feeding episode (Hammerman and Kaplan 1995), there may be another explanation.

Infants with Bronchopulmonary Dysplastic (BPD), who already have breathing problems, encounter even more respiratory challenges when feeding. Timms et al (1993) found that, by increasing respiratory drive through artificially elevating the amount of carbon dioxide in inspired air while feeding, preterm infants significantly decreased their sucking and swallowing rates. This suggested that the increased respiratory drive resulting from carbon dioxide chemoreceptor activation had a detrimental effect on feeding and hence calorific intake. This would imply that BPD infants, who tend to have higher respiratory drives as a consequence of their diminished lung function, may find feeding is inhibited. This can lead to poor growth and development and even feeding aversions.

This analysis will look at the pattern and the relationship of the mean sucking durations and breathing depths and durations for healthy term infants, and see if the feeding strategies adopted by preterm infants, classified as having BPD, are similar. Breath durations will be used to determine how much ventilatory compromise there is for both groups, during sucking and pausing in the intermittent phase of feeding. Although the thoracic band used to measure ventilation does not give a reliable indication of tidal volume, it does show how chest expansion (i.e. depth of breathing) changes during a testing session. It also gives a very reliable measure of breath length, indicating the duration of inhalation and exhalation. This analysis will only look at the intermittent stage of
feeding, so that a direct contrast can be drawn between sucking and pause periods.

6.2 CALCULATION OF MEASUREMENTS

A sucking period was classified as a series of sucks lasting for more than 4 seconds, but less than 20 seconds, and a pause period was characterised as being a time when the infant did not suck for 4 to 20 seconds. Sucking pressure and breathing traces recorded during a feeding session, were displayed together on the screen, using Kaleidagraph, a graphics software package, to identify where sucking periods and pauses took place (see Fig. 6.1).

Figure 6.1
An example of the breathing pattern shown by a bottle feeding term infant during a sucking period and a pause period.

The relevant sucking and breathing data for either a sucking period or a pause was selected. Using the masking option in Kaleidagraph, sucks
registering a pressure of less than 25 mmHg were excluded. Care was also taken to exclude spurious troughs that were not obviously incorporated in the surrounding sucking pattern. A macro, written in the Kaleidagraph application, was used to identify the times at which these consecutive sucking pressure troughs occurred. It did so by looking for the minimum pressure point (i.e. the trough) and returning the corresponding time at that value. A similar macro was used for the breathing data which isolated the time when both the peak (peak inhalation) and the trough (peak exhalation) occurred. In conjunction with giving the time when each event happened, this macro also gave the corresponding values of the depth of the breath at that point (see Fig 6.2). Again vigilance was exercised to exclude bogus peaks and troughs. All of the values representing sucking duration and breath depth and duration, were transferred to an Excel table.

**Sucking Duration**
Total suck duration was defined as the time taken to go from a sucking trough to the next sucking trough. Values greater than 1.8 seconds were discounted as being too long for a normal suck.

**Breathing Depth**
The first trough (indicating the start of inspiration) in the sucking period or pause was used as a starting point (see Fig. 6.2). The value at this trough was subtracted from the value at the following peak (which indicated the end of inspiration and the start of expiration) to give a measure of the depth of inhalation. That peak value was then subtracted from the next trough value to give a measure of the depth of exhalation (see Fig. 6.2). The depths of inhalation and exhalation were each treated
as positive values (i.e. the signs were ignored). The inhalation depth added to the following exhalation depth was taken as a depth measure for the total depth of a breath.

Figure 6.2
A recorded breathing trace showing the points of peak inhalation and peak exhalation, along with measures of breath depth and duration.

**Breath Duration**

The duration of inhalation (like the depth of inhalation) was the time taken from a first trough to the next peak. The duration of exhalation was the time from a peak to the next trough (see Fig. 6.2). The total duration of a breath was taken as the sum of an inhalation duration and the following exhalation duration.

Means and standard deviations were calculated for the depth and duration of a breath in the sucking and pause phases separately. The procedure was identical for both the term and preterm infants. In order
to investigate what was happening to the breathing patterns during intermittent feeding, this analysis will focus on trying to answer different questions that emerged when observing the recorded feeding traces. A trend for feeding behaviour will be established first of all for term infants, and then for the preterm infants with BPD.

6.3 BREATHING REGULARITY

Is breathing more regular during the pause periods than the sucking periods?

One of the first observations from the recorded traces was the different breathing tactics the infant used, depending on whether it was sucking or pausing. During the pause period the breathing pattern appeared to be more orderly and consistent than the breathing style adopted when sucking (see Fig. 6.1). To examine this apparent discrepancy in regularity further, coefficients of variation were calculated for both the breath duration data and the breath depth data, for the sucking and pause periods. (As mentioned in Chapter 4.4, a coefficient of variation is calculated by dividing the standard deviation by the mean, giving a standard measure of the relative variability with respect to the mean for a given parameter. The closer the coefficient of variation is to zero then the more consistent the performance was.)

As can be seen from Figure 6.3, the overall coefficients of variation are smaller for the breath duration data compared to the breath depth data. This would suggest that the infant adjusts ventilation to meet changing respiratory needs by breathing deeper rather than breathing faster. With
respect to the suck and pause periods, smaller coefficients of variation are evident in the pause period for both the duration and depth of the breaths, indicating less relative variability.

**Figure 6.3**
Boxplots showing the difference in the relative variability (coefficient of variation) of the breath duration and the breath depth, for the sucking and pause periods. All 12 infants are included in each boxplot, with the middle thick black line indicating the median value. The upper and lower limits of the boxes show the 25th and 75th percentiles, with the outer hinges marking the last values, falling within an acceptable range.

F-ratios were used to test the significance of the regularity during the pause period compared to the sucking period and were calculated by dividing the variance for the sucking period (hypothesised to be the larger of the two) by the variance for the pause period. It was predicted that the F-ratio would be significantly greater than 1. The corresponding significance levels for the calculated F-ratios for all twelve term infants are given in Table 6.1.
From Table 6.1 it would appear that in the pause period 10 out of the 12 infants have a breath duration that is significantly more regular in the pause period than in the sucking period.

Table 6.1.
The calculated $F$ ratios for breath duration and breath depth, comparing the variances for the sucking and pause periods.

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Breath Duration</th>
<th>Breath Depth</th>
</tr>
</thead>
<tbody>
<tr>
<td>S1</td>
<td>$F(103.91) = 3.16$ **</td>
<td>$F(103.91) = 1.61$ **</td>
</tr>
<tr>
<td>S2</td>
<td>$F(160.93) = 3.98$ **</td>
<td>$F(160.93) = 3.24$ **</td>
</tr>
<tr>
<td>S3</td>
<td>$F(122.85) = 7.66$ **</td>
<td>$F(122.85) = 0.32$ ns</td>
</tr>
<tr>
<td>S4</td>
<td>$F(89.48) = 1.76$ *</td>
<td>$F(89.48) = 2.00$ **</td>
</tr>
<tr>
<td>S5</td>
<td>$F(245.27) = 1.51$ ns</td>
<td>$F(245.27) = 1.39$ ns</td>
</tr>
<tr>
<td>S6</td>
<td>$F(192.31) = 4.99$ **</td>
<td>$F(192.31) = 1.34$ ns</td>
</tr>
<tr>
<td>S7</td>
<td>$F(73.69) = 2.41$ *</td>
<td>$F(73.69) = 1.33$ ns</td>
</tr>
<tr>
<td>S8</td>
<td>$F(207.99) = 3.88$ **</td>
<td>$F(207.99) = 2.36$ **</td>
</tr>
<tr>
<td>S9</td>
<td>$F(85.65) = 1.45$ *</td>
<td>$F(85.65) = 0.62$ ns</td>
</tr>
<tr>
<td>S10</td>
<td>$F(53.65) = 1.78$ **</td>
<td>$F(53.65) = 1.42$ ns</td>
</tr>
<tr>
<td>S11</td>
<td>$F(193.36) = 2.05$ **</td>
<td>$F(193.36) = 0.85$ ns</td>
</tr>
<tr>
<td>S12</td>
<td>$F(125,137)=1.16$ ns</td>
<td>$F(125,137)=1.61$ *</td>
</tr>
</tbody>
</table>

In contrast the variation in the breath depth appears to be less notable, with only 5 out of the 12 infants showing a significant difference in the degree of consistency during the pause period when compared with the sucking period.
6.4 RATE OF BREATHING

Does the mean duration and the mean depth of a breath differ significantly when term infants are sucking when compared to pausing?

In order to see if infants alter their breathing strategy to accommodate swallowing interruptions in the sucking period, t-tests (for unequal numbers) were used to compare the mean breath durations and depths when sucking with mean breath durations and depths when pausing. Each aspect, namely breath duration and breath depth, will be treated separately. The findings of the t-tests (two-tailed) are summarised in Table 6.2 (mean breath duration) and Table 6.3 (mean breath depth).

Table 6.2
A summary of the comparisons of mean breath duration during sucking and pausing.

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>t-values</th>
<th>Subject No.</th>
<th>t-values</th>
</tr>
</thead>
<tbody>
<tr>
<td>S1</td>
<td>t(192)= 0.53 ns</td>
<td>S7</td>
<td>t(140)= -0.62 ns</td>
</tr>
<tr>
<td>S2</td>
<td>t(251)= -2.98 ++</td>
<td>S8</td>
<td>t(304)= -2.68 ++</td>
</tr>
<tr>
<td>S3</td>
<td>t(205)= 3.38 **</td>
<td>S9</td>
<td>t(148)= 2.31 *</td>
</tr>
<tr>
<td>S4</td>
<td>t(135)= -1.93 ns</td>
<td>S10</td>
<td>t(116)= -1.83 ns</td>
</tr>
<tr>
<td>S5</td>
<td>t(270)= 2.73 **</td>
<td>S11</td>
<td>t(227)= 2.97 **</td>
</tr>
<tr>
<td>S6</td>
<td>t(221)= 1.48 ns</td>
<td>S12</td>
<td>t(260)= 1.43 ns</td>
</tr>
</tbody>
</table>

Breathing during sucking sig. faster
Breathing during pausing sig. faster

* p<0.05
** p<0.01

Table 6.2 shows that there appears to be no obvious pattern to the results, with half the infants showing no significant difference between the mean breath duration in the sucking and pause periods. Four out of the twelve infants did breathe significantly faster during the sucking
period, whereas two out of the twelve infants actually breathed significantly faster during the pause period. It would therefore appear that there is a definite trend towards the breathing being more regular in the pause periods, but not necessarily slower.

From the breath depth results, however, a different story emerges. The majority of infants in this case breathe significantly deeper in the pause periods, when compared to the sucking periods. This would suggest that the infants have good respiratory control whereby they breathe deeper, but not necessarily faster during this time. If sucking was an energetic activity that severely compromised respiration, then it might be expected that the infants would breathe significantly faster and shallower during the pause period, which was not the case.

Table 6.3
A summary of the comparisons of mean breath depth during sucking and pausing.

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>t-values</th>
<th>Subject No.</th>
<th>t-values</th>
</tr>
</thead>
<tbody>
<tr>
<td>S1</td>
<td>$t_{(192)} = 4.4$ **</td>
<td>S7</td>
<td>$t_{(140)} = 4.88$ **</td>
</tr>
<tr>
<td>S2</td>
<td>$t_{(251)} = -8.7$ ++</td>
<td>S8</td>
<td>$t_{(304)} = 1.99$ *</td>
</tr>
<tr>
<td>S3</td>
<td>$t_{(205)} = 12.87$ **</td>
<td>S9</td>
<td>$t_{(148)} = 9.12$ **</td>
</tr>
<tr>
<td>S4</td>
<td>$t_{(135)} = -2.18$ +</td>
<td>S10</td>
<td>$t_{(116)} = 9.47$ **</td>
</tr>
<tr>
<td>S5</td>
<td>$t_{(270)} = 4.61$ **</td>
<td>S11</td>
<td>$t_{(227)} = 2.54$ *</td>
</tr>
<tr>
<td>S6</td>
<td>$t_{(221)} = 6.42$ **</td>
<td>S12</td>
<td>$t_{(260)} = 2.17$ *</td>
</tr>
</tbody>
</table>

Breathing during pausing sig. deeper
* $p<0.05$
** $p<0.01$

Breathing during sucking sig. deeper
+ $p<0.05$
++ $p<0.01$
6.5 BREATHING WHEN SUCKING

Is there a 1:1 relationship between sucking and breathing?

A large proportion of the feeding literature discusses the importance of a 1:1 relationship between sucking and breathing. However when scrutinising the recorded traces it appeared that in the majority of cases the duration of a suck was considerably shorter than the duration of a breath. This observation is highlighted in Figure 6.4, where the mean values for suck durations are in general less than the mean values for breath durations during the intermittent feeding period. Also the standard deviations, represented by the bars, tend to be smaller for the sucking durations.

Figure 6.4
Mean suck and mean breath durations for twelve term infants.
Individual t-tests were again carried out for each subject to test the difference between the mean sucking durations and the mean breathing durations during the intermittent sucking phase (see Table 6.4 below).

Table 6.4. Summary of t values, degrees of freedom and p values for the comparison of mean sucking durations and mean breathing durations.

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>t-values</th>
<th>Subject No.</th>
<th>t-values</th>
</tr>
</thead>
<tbody>
<tr>
<td>S1</td>
<td>t(199)=6.24 ***</td>
<td>S7</td>
<td>t(145)=0.64 ns</td>
</tr>
<tr>
<td>S2</td>
<td>t(413)=11.6 ***</td>
<td>S8</td>
<td>t(395)=1.81 *</td>
</tr>
<tr>
<td>S3</td>
<td>t(232)=0.0 ns</td>
<td>S9</td>
<td>t(180)=3.02 **</td>
</tr>
<tr>
<td>S4</td>
<td>t(199)=8.49 ***</td>
<td>S10</td>
<td>t(173)=1.79 *</td>
</tr>
<tr>
<td>S5</td>
<td>t(521)=5.36 ***</td>
<td>S11</td>
<td>t(465)=10.18 ***</td>
</tr>
<tr>
<td>S6</td>
<td>t(371)=1.36 ns</td>
<td>S12</td>
<td>t(289)=7.27 ***</td>
</tr>
</tbody>
</table>

Mean sucking duration sig. faster than mean breathing duration
* p<0.05       ** p<0.0005       *** p<0.0001

As can be seen from the table, nine out of the twelve infants, sucked significantly faster than they breathed during the intermittent phase of their recorded feed. Seven of these nine infants had t values that were extremely significant at p<0.0005. There was no significant difference for the other three infants with S6's mean breath duration actually being shorter than the mean suck duration.

6.6 CONCLUSIONS - TERM INFANTS

From the questions asked above and the subsequent summary statistics, it can be concluded that in the majority of cases the duration of a breath is significantly more consistent during the pause period than the sucking period. This finding would imply that the continuous swallowing in the sucking period prevents a regular temporal breathing pattern being
established. In contrast to the breath duration, the breath depths do not vary significantly in their regularity between the sucking and pause periods.

Bamford (1992) reported a significant decrease in both tidal volume and breathing in newborn infants when sucking. In this study mean breath depths, which could be linked to tidal volume, were also significantly less in the sucking period when compared to the pause period. In contrast the mean breath durations did not appear to exhibit a significant trend in any one direction. From these results it would appear that the infants were able to modulate the depth of a breath, without altering the overall frequency.

With respect to the relationship between sucking and breathing, the majority of the term infants tested sucked at a significantly faster rate than they breathed, whilst feeding in the intermittent phase. These findings do not support those of (Bu'Lock, Woolridge et al. 1990), who implied that it was advantageous for infants to adopt a 1:1 ratio for sucking and breathing. Instead it would seem that sucking is the dominant component of feeding that requires frequent swallowing, which in turn compromises the regularity of breathing. The ventilatory system in the term infants tested appears to be suitably flexible so that breathing can be easily adapted to meet the changing demands of feeding.

6.7 PRETERM INFANTS WITH BPD

Preterm infants with bronchopulmonary dysplasia have, as mentioned in Chapter 1.10., problems with maintaining adequate respiration during
feeding. These problems are thought to stem from the reduced lung capacity and the poor ventilatory response to decreasing oxygen levels in the blood.

Due to the broad nature of the criteria used to clinically diagnose BPD, different infants, within the same category, will need varying degrees of oxygen supplementation. The severity of BPD can be crudely judged by the level of oxygen administered clinically through nasal cannula. The 6 preterm infants tested are categorised in this way in Table 6.5.

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>1st test</th>
<th>2nd test</th>
<th>3rd test</th>
<th>4th test</th>
<th>Class</th>
</tr>
</thead>
<tbody>
<tr>
<td>P1</td>
<td>&lt;0.11</td>
<td>&lt;0.11</td>
<td>&lt;0.11</td>
<td>&lt;0.11</td>
<td>mild</td>
</tr>
<tr>
<td>P2</td>
<td>&lt;0.11</td>
<td>&lt;0.11</td>
<td>&lt;0.11</td>
<td>&lt;0.11</td>
<td>mild</td>
</tr>
<tr>
<td>P3</td>
<td>&lt;0.11</td>
<td>&lt;0.11</td>
<td>&lt;0.11</td>
<td>&lt;0.11</td>
<td>mild</td>
</tr>
<tr>
<td>P4</td>
<td>0.21</td>
<td>0.41</td>
<td>0.51</td>
<td>0.51</td>
<td>severe</td>
</tr>
<tr>
<td>P5</td>
<td>0.125</td>
<td>&lt;0.11</td>
<td>&lt;0.11</td>
<td>&lt;0.11</td>
<td>mild</td>
</tr>
<tr>
<td>P6</td>
<td>0.351</td>
<td>0.51</td>
<td>0.51</td>
<td>no test</td>
<td>severe</td>
</tr>
</tbody>
</table>

6.8 PRETERM BREATHING REGULARITY

Do preterm infants breathe more regularly during the sucking period than the pause period?

Initial observations of all the preterm breathing and sucking traces indicated a more disorganised pattern in both the sucking and pause periods. The striking regularity that was seen when term infants were breathing in the pause period did not appear to be so obvious. To draw a
direct comparison with the term performance, coefficients of variation for
breath duration and depth were calculated as before for the sucking and
the pause periods. All the test sessions for each preterm infant are
displayed in Figure 6.5.

Figure 6.5. Boxplots illustrating the range of the coefficients of variation
across all the test sessions, for breath duration and breath depth, in the
suck and pause periods.
The median value of the coefficients of variation, represented by the middle black line in the boxplots, appears to be more similar in both the suck and pause periods for breath duration and breath depth than the terms (see Fig. 6.3). Preterms P2 and P3, both classified as having mild BPD are exceptions to this rule, showing more inclination towards the characteristics displayed by the terms, namely a marked difference between the two medians in the sucking and the pause periods.

Again the corresponding F-ratios were calculated, and as in the analysis of the term infants, it was predicted that the pause periods would show significantly more regularity, therefore less variance, than the sucking periods (see Table 6.6. for a summary of results).

*Table 6.6. A summary of the level of significance of the F-ratios for all the preterm infants in each of the test sessions.*

<table>
<thead>
<tr>
<th>Subject</th>
<th>Breath Duration</th>
<th>Breath Depth</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1st</td>
<td>2nd</td>
</tr>
<tr>
<td>P1 mild</td>
<td>+</td>
<td>ns</td>
</tr>
<tr>
<td>P2 mild</td>
<td>**</td>
<td>*</td>
</tr>
<tr>
<td>P3 mild</td>
<td>**</td>
<td>ns</td>
</tr>
<tr>
<td>P5 mild</td>
<td>ns</td>
<td>*</td>
</tr>
<tr>
<td>P4 sev.</td>
<td>ns</td>
<td>ns</td>
</tr>
<tr>
<td>P6 sev.</td>
<td>**</td>
<td>ns</td>
</tr>
</tbody>
</table>

Sucking sig. more variation
* = p<0.05
** = p<0.01
ns = not significant

Pausing sig. more variation
+ = p<0.05
++ = p<0.01

83
Preterms P2 and P3 exhibited a pattern of breathing that was most like the term infants by the fourth test session, namely the variation for the depth of a breath was not significantly different for the pause and sucking periods, but the duration of a breath was $F(77,48) = 2.62; p<0.01$ and $F(78,90) = 1.7; p<0.05$ respectively). Preterm P5 likewise showed significantly more regularity for the duration of a breath by the fourth test session ($F(153,67) = 1.72; p<0.01$) but also for the depth of a breath ($F(153,67) = 1.56; p<0.05$).

Preterm P1 showed no significant regularity for either breath duration or breath depth in any of pause periods for the second, third and fourth test sessions. In the first session however, breath duration was found to be significantly more regular in the sucking period ($F(176,210) = 1.58; p<0.05$) and the breath depth significantly more regular in the pause period ($F(176,210) = 2.18; p<0.01$). Preterm infants P4 and P6 classified as having severe BPD showed a rather more varied pattern. P4 showed significantly more regularity in both breath depth and breath duration when pausing in the third testing session only ($F(436,269) = 2.73; p<0.01$ and $F(436,269) = 1.4; p<0.05$ respectively), whereas P6 showed significant regularity for breath depth in the second and third test sessions ($F(67,37) = 2.2; p<0.01$ and $F(158,81) = 1.44; p<0.05$) and breath duration in the first test session ($F(251,178) = 2.4; p<0.01$).

A possible explanation for why some of these preterm infants with BPD do not exhibit a similar breathing pattern to the term infants may be the concentration of oxygen in the blood. Although the infants are being supplemented with oxygen, the additional respiratory demands associated with feeding can cause the oxygen saturation levels in the
blood to fall. Some of these infants may experience lower levels of oxygen saturation when feeding and may need to use the pause periods to breathe more erratically to overhaul the oxygen deficit in the blood. Figure 6.6 shows the range of the mean percentage $O_2$ saturations across all the test sessions as measured by the pulse oximetry, for the individual preterms and for all the terms together. The median value (characterised by the middle black line in the boxes below) for the terms is the highest at 96.8% $O_2$. The two infants with severe BPD not surprisingly have the lowest median saturations at 92.3% for P4 and 89.4% for P6. This may explain the slightly more erratic breathing pattern. The medians for the other preterms are much higher, falling within the limits set by the terms. In fact P3 has a median oxygen saturation value that is the same as the term infants. A higher level of oxygen in the blood would mean less of a need to use the pause period to compensate for an oxygen deficit, thereby showing a similar breathing pattern to the terms, as was the case for P2 and P3.

Figure 6.6.
The boxplots for the individual preterm infants and all the terms showing the range of the mean % of $O_2$ recorded in the different test sessions.
6.9 PRETERM BREATHING RATES

Do infants with BPD breathe faster during sucking or pausing?

A manifestation of the respiratory difficulty infants with BPD experience when feeding is their tendency to pant during the pause periods. A lack of ventilatory reserves and the periods of apnoea that tend to appear during the sucking period contribute to the oxygen deficits that need to be overhauled in the pause period. Pause periods therefore allow the infant to compensate for this deficiency by substantially increasing the frequency of breathing. It would therefore be expected that if an infant is using the pause period to increase oxygen concentrations, a significant decrease in breath duration would be expected during the pause period (see Fig. 6.7).

It appears that for all the infants with mild BPD the mean breath durations during pausing are significantly shorter than the mean breath durations during sucking in at least one of the first two test sessions (see Fig. 6.7 and Table 6.7).

By the third and fourth testing sessions the pattern has changed for all the infants, apart from P5 in the third test, with the mean breath durations being no longer significantly shorter in the pause period. Some of the infants showed a switch whereby the mean breath durations in the sucking phase were shorter than the mean breath durations during the pause phases (see Table 6.7).
The two infants with more severe BPD (P4 and P6), displayed a significant degree of panting in the first 3 test sessions, as evidenced by the large difference between the mean breath durations when pausing and the mean breath durations when sucking (see Fig. 6.7 and Table 6.7).
Table 6.7. Results of t-tests, comparing the mean breath duration when sucking with the mean breath duration when pausing for the 6 preterm infants with BPD.

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>1st test</th>
<th>2nd test</th>
<th>3rd test</th>
<th>4th test</th>
</tr>
</thead>
<tbody>
<tr>
<td>P1 mild</td>
<td>t(384)=2.79 **</td>
<td>t(341)=2.14 *</td>
<td>t(400)= -1.1</td>
<td>t(289)= 0.47</td>
</tr>
<tr>
<td>P2 mild</td>
<td>t(181)=0.38 **</td>
<td>t(171)=2.96 *</td>
<td>t(102)= 0.5</td>
<td>t(123)= 0.19</td>
</tr>
<tr>
<td>P3 mild</td>
<td>t(147)=3.17 **</td>
<td>t(141)= -1.6</td>
<td>t(110)= -0.9</td>
<td>t(148)= 1.35</td>
</tr>
<tr>
<td>P5 mild</td>
<td>t(391)=2.26 *</td>
<td>t(176)= -0.5</td>
<td>t(660)= 3.67 ***</td>
<td>t(218)= -2.1 +</td>
</tr>
<tr>
<td>P4 sev.</td>
<td>t(321)=4.4 ***</td>
<td>t(612)=5.11 ***</td>
<td>t(703)= 4.56 ***</td>
<td>t(227)= -0.6</td>
</tr>
<tr>
<td>P6 sev.</td>
<td>t(427)=3.6 ***</td>
<td>t(102)= 2.07 *</td>
<td>t(237)= 2.8</td>
<td>no test</td>
</tr>
</tbody>
</table>

Breathing during pausing sig.  Breathing during sucking sig. faster
*** p < 0.001
**  p < 0.01
*   p < 0.05

Nevertheless P4 did improve, so that by the 4th test session, breathing while pausing was actually slower than breathing when sucking. The severity of BPD can therefore be clearly seen in the different breathing patterns that emerge in the sucking and pausing periods of intermittent feeding.

**6.10 PRETERM BREATHING WHEN SUCKING**

Are mean suck durations significantly shorter than mean breath durations for infants with BPD?

The observed feeding pattern for 9 of the 12 term infants tested, indicated that when sucking, the mean suck durations were significantly
shorter than the mean breath durations during that period, suggesting that sucking was the dominant feature in the feeding process.

Figure 6.8. Preterm mean suck durations and the corresponding mean breath durations, across the different testing sessions.

Figure 6.8 shows the corresponding values for each of the individual preterms and how this pattern developed across the 4 testing sessions.
In the first test preterms P1 and P5 sucked significantly slower than they breathed, but in the later testing sessions this difference was no longer significant as the suck durations decreased in relation to breath durations (see Table 6.8). Preterm twins P2 and P3 sucked faster than they breathed throughout all the testing sessions. These differences were significant for P3 in all the sessions, and in all but the first session for P2 (see Table 6.8). Again these two infants were displaying term features in their feeding behaviour.

Table 6.8
Results of t-tests (2-tailed) comparing the mean suck durations with the mean breath durations across all 4 test sessions.

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>1st test</th>
<th>2nd test</th>
<th>3rd test</th>
<th>4th test</th>
</tr>
</thead>
<tbody>
<tr>
<td>P1 mild</td>
<td>t(383)=-5.33</td>
<td>t(507)=0.69</td>
<td>t(467)=1.05</td>
<td>t(-0.55)=-0.5</td>
</tr>
<tr>
<td></td>
<td>+++</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P2 mild</td>
<td>t(174)=1.02</td>
<td>t(224)=5.52</td>
<td>t(131)=2.75</td>
<td>t(174)=7.66</td>
</tr>
<tr>
<td></td>
<td>***</td>
<td>**</td>
<td>***</td>
<td>***</td>
</tr>
<tr>
<td>P3 mild</td>
<td>t(136)=2.79</td>
<td>t(131)=1.97</td>
<td>t(161)=4.92</td>
<td>t(168)=3.46</td>
</tr>
<tr>
<td></td>
<td>**</td>
<td>*</td>
<td>***</td>
<td>***</td>
</tr>
<tr>
<td>P5 mild</td>
<td>t(668)=-2.15</td>
<td>t(314)=-0.6</td>
<td>t(929)=0.0</td>
<td>t(297)=-0.73</td>
</tr>
<tr>
<td></td>
<td>+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P4 sev.</td>
<td>t(341)=2.02</td>
<td>t(355)=1.35</td>
<td>t(822)=-2.01</td>
<td>t(309)=3.14</td>
</tr>
<tr>
<td></td>
<td>*</td>
<td></td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>P6 sev.</td>
<td>t(446)=-4.51</td>
<td>t(123)=-0.4</td>
<td>t(298)=-1.02</td>
<td>no test</td>
</tr>
<tr>
<td></td>
<td>+++</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Sucking significantly faster  Breathing significantly faster

*     p<0.05  +     p<0.05
**    p<0.01  ++    p<0.01
***   p<0.005 +++   P<0.005

The two infants with severe BPD (P4 and P6), exhibited a more varied pattern. Preterm P4 showed a significantly shorter sucking duration in the 1st and 4th test, but not in the 2nd or 3rd test. In fact test 3 showed
that mean breath durations were actually significantly shorter than mean suck durations. A similar finding was observed for P6 in the first test. Tests 2 and 3 showed the same pattern but with the difference between the means not being significant.

6.11 CONCLUSIONS - INFANTS WITH BPD
In conclusion, it can be seen that some of the infants diagnosed as having mild BPD started to show some of the significant breathing duration regularity exhibited by the terms during the pause period of feeding. The infants with severe BPD failed to show any uniformity during the pause period in any of the test sessions. This could be explained by the fact that these infants experienced much lower concentrations of oxygen throughout feeding sessions, indicating that their respiration was being severely compromised. The pause periods may therefore be more disorganised to try and overhaul these deficiencies. To reinforce this the mean breath durations during this period were also significantly shorter, indicating that the infant was panting to try and increase the level of oxygen in the blood.

With regards to the mean suck duration and the relation to mean breath duration, P2 and P3 showed significantly shorter suck durations like the terms, in at least 3 of the 4 testing sessions. Some of the preterms (P1, P4 and P6) were breathing significantly faster than they sucked in one of the test sessions. These findings suggest that the difficulty breathing and the resulting higher concentrations of CO₂ inhibited the sucking from dominating the feed. A similar outcome was reported by Timms, DiFiore et al. (1993) where sucking frequency was significantly reduced in preterm infants when the respiratory drive was heightened artificially.
by increasing the concentration of CO\textsubscript{2} in the air inhaled. They suggested that there were perhaps neural mechanisms that suppressed the motor output needed for sucking. This may help explain why infants with severe BPD, like P6, have such difficulty feeding.

6.12 SUCKING RATES

Previous research has found that as term infants increase in gestational age, the number of sucks per second when feeding nutritively also increases (Wolff 1968; McGowan, Marsh et al. 1991). Comparable findings have likewise been reported for non-nutritive sucking while sleeping in the case of preterm infants (Hack, Estabrook et al. 1985). Similar trends were observed in this data set, with mean sucking durations for the five term infants tested again at 3 weeks of age, decreasing (see Fig. 6.9). Four out of five of these decreases were very significant (see Table 6.9).

Figure 6.9

a. Mean suck durations for 5 of the 12 term infants, tested in their second day and again at 3 weeks of age. (Subject numbers correspond to those used previously in this chapter). Also mean suck durations for the six preterm infants tested in their first week of feeding and again 3 weeks later (2 weeks in the case of P6).
All the preterms (except P6 who had severe BPD), decreased their mean suck duration over the 3 week period (see Fig. 6.9). For preterms P1, P2 and P5 with mild BPD these decreases were significant. Although P3 did not show a decrease in mean suck duration that was significant, this could be explained by the fast mean suck duration (0.78 sec.) already attained in the first test.

Table 6.9
A summary of the term and preterm t-tests (one-tailed) comparing the mean suck duration for the first test with the mean suck duration for the final test.

<table>
<thead>
<tr>
<th>Term</th>
<th>t values</th>
<th>Preterm</th>
<th>t values</th>
</tr>
</thead>
<tbody>
<tr>
<td>S5</td>
<td>t(230)= 4.03 ***</td>
<td>P1</td>
<td>t(324)= 6.1 ***</td>
</tr>
<tr>
<td>S6</td>
<td>t(350)= 0.88</td>
<td>P2</td>
<td>t(189)= 4.34 ***</td>
</tr>
<tr>
<td>S8</td>
<td>t(263)= 5.9 ***</td>
<td>P3</td>
<td>t(158)= 1.29</td>
</tr>
<tr>
<td>S11</td>
<td>t(322)= 10.41 ***</td>
<td>P4</td>
<td>t(459)= 1.64</td>
</tr>
<tr>
<td>S12</td>
<td>t(562)= 6.77 ***</td>
<td>P5</td>
<td>t(319)= 1.72 *</td>
</tr>
<tr>
<td></td>
<td></td>
<td>P6</td>
<td>t(337)= -0.98</td>
</tr>
</tbody>
</table>

Sucking sig. faster in last test
* p<0.05
*** p<0.0005

The mean suck durations for the two infants with severe BPD did not decrease significantly. Preterm P4 was just outside the critical level of significance (p=0.05), whereas the mean suck duration for P6 actually increased. Again it might be worth reinforcing that this was the third and last test for P6, as feeding problems worsened and milk was then ingested solely through nasogastric tubes.
6.13 CONCLUSIONS - SUCKING RATES

Sucking durations appear to decrease over time. There does, however, appear to be substantial variation between the actual duration recorded for all the infants within both groups. For instance some of the preterm infants, that were of a lower chronological age, were able to suck as fast if not faster than the term infants. The results cannot therefore be explained solely by increases in gestational age but more by the coexistence of neural maturation and feeding experience.
Sucking in the neonate is a highly evolved skill that is often misconstrued as being a purely reflexive behaviour over which the infant exercises little or no control. Two different modes of neonatal sucking, namely nutritive and non-nutritive, were highlighted by Wolff (1968). Nutritive sucking was seen as an unstructured behaviour concerned with the extraction of milk from the breast or bottle. In contrast non-nutritive sucking, that was present in the absence of milk flow, was observed in both sleep and active states. The richness of non-nutritive sucking as an early behaviour was illustrated when infants learned to control the focus of a projected visual display through adjusting their sucking rhythm (Kalnins and Bruner, 1973).

Although there is evidence of in utero non-nutritive sucking (Ianniruberto and Tejani 1981), the infant faces a very different challenge when sucking nutritively. At birth the infant has had no previous experience controlling the flow of milk and co-ordinating swallows and breaths, but twenty-four hours post partum the neonate performs these tasks proficiently.

This study proposed that nutritive sucking should be seen as a precocious motor skill requiring the integration of internal information, about the oral movements which generate changes in pressure, with information concerning the subsequent flow of milk from the feeding vessel.
7.1 SUCKING CONTROL

Good oral motor control is not only essential for feeding but also communication. Sucking control and speech production are dependent on intact oral motor musculature and the relevant neural mechanisms. The oral region is a highly sensitive area, with tongue, lip and throat movements being represented by a disproportionately large area of the motor cortex (Carlson 1991).

The oral motor region has numerous sensory outlets. Within the mouth, for instance, there is evidence to suggest that afferents of the infraorbital nerve fire in response to changes in intraoral pressures produced during phonation and blowing (Furusawa, Yamaoka et al. 1994). Linked to this, recent studies have found that periodontal mechanoreceptors show great sensitivity to the changing forces exerted on the incisors (Trulsson and Johansson 1996). The position of the tongue and the jaw are also specified by both proprioceptive information and feedback from the muscles, tendons and corollary discharge (Grover and Craske 1992). These different examples illustrate the variety of sensory information that is available within the oral cavity so that oral movements, like those required for sucking, can be finely controlled.

7.2 INTERNAL τ-GUIDE

Wolff (1968), who pioneered research into the temporal organisation of sucking, primarily focused on the temporal aspects of non-nutritive infant sucking. He documented the sucking rhythm of term and preterm infants by quantifying the intersuck interval at different gestational ages. Although temporal differences were found, the limitations of looking solely at these quantitative changes and not using nutritive sucking
which would incorporate more temporal variability to control the flow of milk, were recognised.

The analysis of nutritive sucking control in this thesis has investigated the temporal control within a suck, rather than between sucks. This mode of analysis has provided much more information about the actual form of an individual suck and how the pressure changes are controlled. The internal \( \tau \)-guide model proposed an internal dynamic prototype that provides temporal information for movements to couple onto. Jeannerod (1994) emphasised the importance of internal representations when trying to understand how movements are controlled.

Any study dealing with motor behaviour must take into account the fact that the overt component (the movement) is only part of the phenomenon as a whole. The hidden component (the representation) exists in its own right. Jeannerod (1994)

The very high \( r^2 \) values found in this study, indicated a strong coupling between an internal \( \tau \)-guide and the \( \tau \) of the intraoral sucking pressure, for both the increasing and decreasing suction phases. Similar strong couplings have also been found for adult subjects performing a hand to mouth task (Lee, Craig et al. 1997). These preliminary studies suggest that perhaps the internal representation of a given movement is specified by a constantly accelerating \( \tau \)-guide.

The corresponding \( k \) values for this study have also provided interesting information about the shape of the pressure velocity profiles. The increasing suction phase was characterised by a velocity profile that had an abrupt approach to the destination (peak negative pressure). This suggested that the infants were trying to maximise the impetus of the
pressure generated intraorally. The decreasing suction phase, on the other hand, displayed velocity profiles that had a much more gradual deceleration. In this case the infants were slowing the movement of the milk to the back of the mouth, thereby, minimising the danger of it coming into contact with the trachea.

In conclusion the resulting $r^2$ and $k$ values from the internal $\tau$-guide model appear to provide a wealth of information about the form of a movement and how it is controlled.

7.3 **PRETERM SUCKING DEFICITS**

A disproportionate number of infants born at a low gestation and birthweight develop some form of motor deficiency (Volpe 1994). Wolff (1968) proposed that non-nutritive sucking rhythms may be able to give clinicians some idea of an infant's neurological status. Infants who suffered from some form of perinatal stress were found to display sucking rhythms that were significantly different from the normal pattern. No correlation was made, however, between specific abnormalities in a sucking rhythm and a particular clinical diagnosis. One limiting factor was the way sucking rhythms were found to change according to a particular behavioural state. In this study however, the internal $\tau$-guide model provided a more reliable means for examining the temporal structure of preterm sucking movements, and predicting future motor function.

Differences in the strength of coupling were found for the majority of the six preterm infants tested who were all born at less than 30 weeks gestation and 1400g birthweight. As each infant's clinical history
differed, so did the corresponding couplings between the internal τ-guide model and the τ of the intraoral pressure. Follow up physiotherapy motor assessments at 6 months corrected age showed that the four infants tested were showing some form of motor delay, with the infant with the weakest coupling showing the greatest delay. It would therefore appear that the internal τ-guide model, when applied to sucking, does give some indication of the infant's future motor outcome. More testing and subsequent follow ups would be needed to draw more definitive conclusions about the findings.

7.4 BREATHING REGULATION
In addition to controlling sucking pressures, infants must be able to maintain an adequate level of ventilation throughout the feeding episode, by coordinating breathing with sucking. The term infants tested did so effortlessly by adapting the regularity of the breath duration and modifying the breath depth during the sucking phase. The pause period differed from the sucking period in that the breathing pattern was characterised by significantly more regular breath durations and significantly greater breath depths. Preterm infants classified as having BPD, not surprisingly, experienced more difficulty coordinating breathing with sucking. Nevertheless by the fourth week of testing some of these infants showed similar patterns to the term infants. The two infants with severe BPD, that had noticeably lower concentrations of oxygen in their blood during feeding, differed by breathing at a significantly faster rate in the pause periods to try and override the oxygen deficiency. These breathing difficulties could make feeding problematic and uncomfortable and may even inhibit the development of a sucking rhythm (Timms, DiFiore et al. 1993).
7.5 CONCLUSIONS

Feeding provides the first instance where a neonate can actively interact with its environment by changing the intraoral pressure to modify the flow of milk from the bottle or breast. Feeding also presents the first opportunity for the infant to communicate with caregivers. During a feeding episode the infant commands the attention of the caregiver, who frequently engages in communicative behaviours such as stroking or talking. Kaye (1977) has suggested that the observed pause periods in the intermittent feeding period present an early instance where the caregiver and infant can learn the bases of turn taking. The importance of sucking behaviour and postnatal communication have been emphasised in a study concerned with enhancing mother-infant bonding in Merino ewes (Nowak 1996).

Feeding can therefore be seen as a very rich behaviour that provides a neonate with the first opportunity of interacting with the surrounding physical and social environment.
REFERENCES


APPENDIX I

COMPUTING THE $\tau$ COUPLINGS

INTERNAL $\tau$-GUIDE

The hypothetical constant acceleration $\tau$-guide has a single parameter, the time $T$ it takes to reach its goal. Imagine the guide as an object starting from rest at time $t=0$, accelerating at a constant rate $A$ and reaching its goal after time $T$. Thus its starting distance from the goal is $\frac{1}{2}AT^2$. After time $t$ it will have travelled a distance $\frac{1}{2}At^2$ and so its distance from the goal will be $\frac{1}{2}AT^2 - \frac{1}{2}At^2$. Its velocity then will be $At$. Therefore, the value of $\tau$-guide at time $t$ will be $\frac{1}{2}AT^2 - \frac{1}{2}At^2 / (At)$ ($\tau$ is conventionally taken as being negative during approach to a goal). Thus, simplifying

$\tau$-guide at time $t = \tau_g(t) = \frac{1}{2}(t-T^2/t)$

for $0 < t < T$.

The hypothesis is that at any time $t$ ($0 < t < T$) the observed $\tau$, $\tau_0(t)$, is coupled to $\tau$-guide, $\tau_g(t)$. That is

$\tau_0(t) = k\tau_g(t) = \frac{1}{2}k(t-T^2/t)$

for some constant $k$.  

111
FORMULA FOR CONSTANT K

The value of $k$ that yields the best fit on the least sum of squares criterion is computed as follows. Let $S$ be the sum of the squares of the deviations of the observed values, $\tau_0(t)$, from the values, $k\tau_g(t)$, predicted by the hypothesis. (The summation is taken over the range $t = t_s$ to $t = t_e$ where $t_s$ is just above zero and $t_e$ is just below $T$. This is to exclude extreme values where measurement error is higher.) Thus

$$S = \sum_{t=t_s}^{t=t_e} [\tau_0(t) - \frac{1}{2} k(t-T^2/t)]^2$$

$S$ is minimum when $\frac{dS}{dk} = 0$, i.e., when

$$- \sum_{t=t_s}^{t=t_e} [\tau_0(t) - \frac{1}{2} k(t-T^2/t)](t-T^2/t) = - \sum_{t=t_s}^{t=t_e} \tau_0(t)(t-T^2/t) + \sum_{t=t_s}^{t=t_e} \frac{1}{2} k(t-T^2/t)^2 = 0$$

Thus, solving for $k$,

$$k = \frac{\sum_{t=t_s}^{t=t_e} \tau_0(t)(t-T^2/t)}{\sum_{t=t_s}^{t=t_e} \frac{1}{2} (t-T^2/t)^2}$$
**Computing the Linear Regression**

The formula for calculating the linear relationship between the $k\tau_g$ and the $\tau_p$, using the least sum of squares model (Hays 1970, p.498), is computed as follows:

\[ r^2 = 1 - \text{sample variance of estimate for standard scores} \]

\[ = 1 - \frac{\sum (\tau_p^* - k\tau_g^*)^2}{N} \]

where

\[ \tau_p^* = \frac{\tau_p - \text{mean}(\tau_p)}{\text{s.d.}(\tau_p)} \]

\[ k\tau_g^* = \frac{\tau_g - \text{mean}(\tau_g)}{\text{s.d.}(\tau_g)} \]

\[ N = \text{number of samples} \]
APPENDIX II

PHYSIOTHERAPIST ASSESSMENT - NOTES

Preterm P4

Postural Reactions - Vojta Scale
7 of 7 postural reactions are presented normally, in the 9-12 month movement quality range.
No. 7 presented in a transition level reaching into the 2nd phase (6/7mth).
No single abnormal response was noticed.
P4’s CNS reflecting a co-ordination level of 9 months or above when provoked by a sudden change of posture.

Milestones achieved:
• rolling over back to front both sides (6mth)
• commander crawling (one-sided over right arm) (9mth)
• crawling knees and hands/rocking forward-backwards in crawling position (10mth)
• transition free sitting to crawling position and back
• scissors grasp bilateral: picks bead between thumb and side of curled index finger thumb; proximal joint extended, distal slightly flexed (8mth)

Other Observations
P4 - mild degree of hypotonia in classic areas seen in preterm infants:
1. shoulder girdle: poor stabilising of scapula via spine (mid thorax) - expressed in crawling position - shoulders poorly depressed.
2. **abdominal muscles**: poor co-contraction - expressed in sitting distended tummy


**Summary:**

P4 is 1 year and 3 weeks old minus 15 weeks to correct age.

At time of assessment her corrected age is 9 months.

Her co-ordination is reaching a maturity level of 9 month and better.

Her gross motor function ranges between 9-10 mth

Her fine motor function is around the 8 mth level.

However the quality of her gross motor function is lacking stability and smoothness due to poor muscular power in certain areas as described above. But there is not functional motor delay as such and she performs age appropriately.
Preterm P6

Postural Reactions - Vojta Scale
5 out of the 7 postural reactions are “normal” at the 6 mth level, when responding to sudden change of posture.
However 2 of the responses showed marked immaturity, namely 1 and 4 (see diagram)
Traction (pull to sit) (1): head struggling (shaky) to hold up, spine not sufficiently extended, marked hypotonic muscular tone around spine and trunk.
Vojta reaction (4): (top arm and leg to be assessed) Right uppermost arm - Moro abnormal/leg alright. Left side arm and leg normal display of 8-9mth level -good.

Milestones Achieved:
Hand function
• track, follow and grasp of rattle (45° off midline) across midline including weight transfer of trunk to the opposite side, right and left good. (5mth)
• holding rattle with both or one hand and putting into mouth
• release of rattle no problem and transfer from right to left and left to right good.
• palmar grasp reflex absent (overridden)
• shoulder depression displayed at a 3mth level.

Other Observations
P6 moderate degree of hypotonic muscular tone - particularly affected is his trunk muscles (abdominal, back and neck muscles). This leads to reinforcing compensation patterns especially elevation of his shoulders (no neck) and retraction of elbows which again prevents skills like stabilising the shoulder girdle in order to push up when lying on his tummy.
Prone is unacceptable as he gets stuck in this position - limits the chances of gaining any proprioceptive and sensory experience to prepare for gross
motor function (crawling) which is linked to good arm and hand function in later in later month is completely inhibited.

Summary
P6 is 9.5 months minus 11 weeks for premature birth. His corrected age is 7 months at the time of assessment. His CNS reflects a co-ordination level of 6 months for reactions to postural changes. Gross motor activity is partly age appropriate (rolling over back to front/6 months) but in other area dramatically delayed (prone at 2 month level). P6’s biggest problem is his poor muscular power in all areas which prevents him achieving any kind of stability against gravity and is reason for his delay.
Preterm P5

Postural Reactions - Vojta Scale
3-4 out of the 7 postural reactions are normal, namely 1, 2, 7 and “5” (see diagrams on sheet), showing a 6 month level of motor quality. In the other 3 postural responses, she shows some stiffening of her legs and clenching of her left hand.

Milestones Achieved.
• grasp of the rattle (45° off midline) bilateral achievement good
• holding rattle with both hands and put into mouth good
• not able to release the rattle
• also not able to transfer rattle from right hand to left hand.
• shoulder depression at 3 month level
• palmar grasp reflex still present in moderate to strong fashion.

Summary
P5 is 9 months minus 13 weeks for preterm birth
Her corrected age is 6 months at the time of assessment.
Her co-ordination of body posture when suddenly changed, is in most cases age appropriate apart from a few occasions where she responds abnormally. In these cases she offers a recurring flexor pattern and a clenched fist on the left side. This reflects some irritability of the CNS which most likely will sort itself out.
No observed hypotonia.
Her gross motor activity and hand function presents on a 3-4 month level.
There is still a degree of palmar grasp reflex bilaterally which should really be gone at this stage. Weight transfer in supine from side to side is poor also the abdominal area is poorly developed in terms of stability.
There is a 2-3 month gap between P5’s motor performance age and her corrected age, suggesting a motor delay.